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REPORT BY THE SPECIAL MEDICAL COMMITTEE INVESTIGATING MATERNAL MORTALITY ON 319 DEATHS ASSOCIATED WITH PREGNANCY AND CHILDBIRTH OCCURRING IN NEW SOUTH WALES, 1950 TO 1956.

THE Special Medical Committee investigating Maternal Mortality was appointed by the Minister for Health in 1939, as part of a campaign for the reduction of maternal mortality in New South Wales. Since that time all maternal deaths in the metropolitan area of Sydney have been investigated. The objects of this investigation are to discover the causes of the deaths and those which were preventible, to establish what were the avoidable factors, if any, presented in the case history, and to advise the Minister for Health on these matters.

As the maternal mortality rates for the "remainder of State" were consistently higher than those for the metropolitan area, the Committee recommended to the Minister for Health that the investigation should be extended to all maternal deaths occurring in New South Wales; extension to the South Coast and Hunter River Health Districts took place during 1952, and to the remainder of the State during 1954. The scope of this report is from 1950 to 1956 inclusive.

The personnel of the Committee during these years has included the following members; the late Emeritus Professor J. C. Windeyer, the late Dr. E. S. Morris, the late Dr. T. Dixon Hughes and Dr. T. H. Small.

The present members are as follows: The Chairman, the Director-General of Public Health Dr. H. G. Wallace; Professor B. T. Mayes; Emeritus Professor F. J. Browne; Dr. J. N. Chesterman, representing the Women's Hospital, Crown Street; Dr. K. J. Howell, representing the Royal Hospital for Women; Dr. E. A. Tivey, representing the British Medical Association; Dr. Grace Cuthbert Browne, Director of Maternal and Baby Welfare and Medical Secretary. Alternates for the representatives have been Dr. R. B. C. Stevenson, Dr. Ida B. Saunders and Dr. Mervyn Elliot-Smith.

The Committee now presents a report of its findings for the seven years 1950 to 1956 inclusive. During this time, a total of 319 deaths were considered, 262 of which were classified as "maternal deaths" and 57 as "non-maternal". Owing to exigencies of space, only 41 of these have been selected for detailed description in this report. The selection has been made so that the cases described may be fairly representative and illustrative of the main groups of diseases, abnormalities or complications that were responsible for the deaths.

The Committee wishes to place on record its appreciation of the valuable cooperation offered by the medical superintendents and other members of the staff of the obstetric hospitals, by obstetric consultants and by general practitioners, which has helped to make the work of the Committee effective. The Committee also wishes to place on record its appreciation of the work of the medical staff of the Division of Maternal and Baby Welfare and other officers of the Department of Public Health cooperating in this investigation.

The Committee believes that it is the strict observance by all concerned of the confidential nature of the information made available that has encouraged the cooperation of the medical profession. No person other than the medical members attends the meetings, and members of the Committee do not at any time retain the material circulated to them before the meetings. Notes on the *modus operandi* of the Committee meetings are set out later in the report.

The Tables I and II set out hereunder indicate the numbers and rates in New South Wales of "maternal" deaths from 1920 to 1956 and of live births and still births from 1937 to 1956 according to the International List of Classifications of Diseases, Injuries and Causes of Death (Section XI relating to deliveries and complications of pregnancy, childbirth and the puerperium). The Committee wishes to point out that deaths from hypertension existing before pregnancy and from renal disease

existing before pregnancy have been included in this Section, although in the sixth Revision of the International Classification there is no provision for them.

The first two columns in Table I indicate the great reduction in maternal mortality which followed the introduction of antibiotics, of modern principles of ante-natal care, of improved obstetric care generally, of the improved social and economic conditions affecting the mother, and other factors. The other columns show the further reductions until the rate became less than one per 1000 live births in 1952.

The Method of Obtaining the Information and of Assessment of the Histories in the Fatal Cases.

On receipt of a notification from the Registrar-General of a death associated with pregnancy or childbirth, tabulated *questionnaires* are sent to the medical practitioner or hospital concerned. These *questionnaires* are devised to provide the Committee with a methodical and routine presentation of the facts. If more than one medical practitioner or hospital is concerned, additional *questionnaires* are sent. The medical staff of the Division of Maternal and Baby Welfare correlates the information and seeks further particulars if necessary, until all the information obtainable is complete.

A typed copy of this correlated case history to be investigated is made available to each member of the Committee before the meeting. At the meeting, assessment is made of the cause of the death with its classification according to international standards, and of the "preventibility" of the death.

The Committee wishes to make it clear that full cognizance is taken of the practical difficulties arising at the bedside of the patient, whether in hospital or not, and whether in Sydney or outside it. The object is to ascertain whether reasonable facilities exist, whether they have been used, whether reasonable care has been exercised by the medical and nursing attendants, whether hospital administration has been at fault, and whether the mother cooperated in seeking early ante-natal care and in following medical advice. It is evident throughout the study that increased health education is necessary to reach those mothers who are either ignorant of or indifferent to the necessity of presenting themselves for ante-natal care early in pregnancy and of following the medical advice given.

"Preventibility" is considered under the headings of "The Primary Avoidable Factor". The primary avoidable factor is the first deviation from a reasonable standard of obstetric practice, or the failure to cooperate on the part of the patient or her relatives. Primary avoidable factors used in assessment of any preventible death are: (i) error of judgement or in management on the part of the doctor; (ii) error of management in hospital; (iii) omission or inadequacy of ante-natal care; (iv) negligence on the part of the patient, or lack of cooperation on the part of the patient and/or her relatives in following medical advice; (v) failure to use reasonable facilities. In many instances "preventibility" is based on more than one of these factors.

Examples of preventible deaths studied by the Committee and classified under these headings will be given later in the text, but a brief explanatory note is given here.

"Error of judgement or in management by the doctor" means, for example, either a mistake in diagnosis with consequent error in treatment, or the correct diagnosis with the wrong treatment, etc.

"Error of management in hospital" is a broad heading, and includes failure by the nursing staff or resident medical officers to report the condition of the patient to the doctor in charge of the case, or failure to carry out instructions, or a combination of errors in judgement or management by several members of the staff of a hospital.

"Omission or inadequacy of ante-natal care" is self-explanatory, and refers only to the doctor in charge of a patient.

"Negligence on the part of the patient or her relatives" is again self-explanatory, and refers not only to the patient's failure to attend for ante-natal care, but also to her failure to follow advice given, including refusal to obey instructions to enter hospital for treatment. The relatives may be at fault in failing to summon medical assistance when needed, or may actively interfere to prevent the doctor's instructions being obeyed.

Table I.

Maternal Mortality Rates in New South Wales, 1920 to 1956, "Maternal" Deaths Only, per 1000 Live Births.

Years 1920 to 1954.	Maternal Deaths per 1000 Live Births.	Years 1943 to 1949.	Maternal Deaths per 1000 Live Births.	Years 1950 to 1956.	Maternal Deaths per 1000 Live Births.
1920–1924	5·39	1943	3·42	1950	1·12
1925–1929	5·79	1944	3·12	1951	1·01
1930–1934	5·93	1945	2·25	1952	0·92
1935–1939	5·30	1946	1·65	1953	0·64
1940–1944	4·23	1947	1·87	1954	0·67
1945–1949	1·69	1948	1·37	1955	0·82
1950–1954	0·87	1949	1·37	1956	0·82

"Failure to use reasonable facilities" has many applications, and refers not only, for example, to failure to summon a mobile blood transfusion unit when needed, or to call in a consultant in a difficult case, or to make use of laboratory services in cases of infection, etc., but also to other factors which have gained in importance with the increasing number of migrants in this country—for example, language difficulties. These are not insuperable, as the Department of Public Health provides translations of specially selected material suitable for expectant mothers.

In the years 1950 to 1956 inclusive, 319 deaths were studied by the Committee; of these 262 were considered to be due to "maternal" causes and 57 to associated diseases—that is, diseases associated with pregnancy but not arising out of it, such as heart disease, tuberculosis, etc. Of the 262 deaths due to "maternal" causes, 159 were considered to be preventible. Deaths regarded as non-preventible were those in which it was concluded that the patient had cooperated satisfactorily with her medical attendant, and had received reasonable medical care.

Investigation of Clinical Histories in Fatal Cases.

Toxæmias.

The clinical histories in 53 cases of maternal death attributed to "toxæmia" during the years 1950 to 1956 were examined by the Committee. These include: (*a*) eclampsia, preeclamptic toxæmia (preeclampsia) and post-partum eclampsia, 33 deaths; (*b*) chronic hypertension with or without superimposed preeclamptic toxæmia, eight deaths; (*c*) chronic glomerular nephritis, four deaths; (*d*) "other" toxæmias, eight deaths.

Preeclamptic Toxæmia and Eclampsia.

Deaths from preeclamptic toxæmia with or without eclampsia are nearly always preventible. Unfortunately, whereas most doctors are now aware of the dangers of toxæmia and, therefore, see their patients regularly, not all realize the significance of its early signs. For example, the doctor may take his patient's blood pressure at regular intervals and test her urine, but may leave her to weigh herself, and by so doing miss an abnormal increase in weight as a sign of impending toxæmia until a rise in blood pressure draws attention to what is occurring. It is of the utmost importance to realize that albuminuria is a late and dangerous sign of toxæmia, and always demands the patient's immediate admission to hospital. Too many doctors are unaware of this fact, and allow a patient to continue ambulant with a "cloud" or a "trace" of albumin in the urine while merely restricting salt intake or prescribing some form of reducing diet.

TABLE II.

Live Births, Still Births and "Maternal" Deaths, New South Wales: Numbers and Rates per 1000 Live Births, 1937 to 1956.

Year.	Live Births.		Still Births.		Total Births, Live and Still.	"Maternal" Deaths.	
	Number.	Rate per 1000 of Mean Population.	Number.	Rate per 1000 Total Births, Live and Still.		Number.	Rate per 1000 Live Births.
1937 ..	47,497	17·62	1452	29·66	48,949	233	4·91
1938 ..	47,319	17·38	1473	30·19	48,792	231	4·88
1939 ..	48,003	17·45	1360	27·55	49,363	200	4·17
1940 ..	49,382	17·78	1342	26·46	50,724	209	4·23
1941 ..	51,729	18·47	1464	27·52	53,193	209	4·04
1942 ..	52,647	18·60	1411	26·10	54,780	209	3·97
1943 ..	57,265	20·04	1465	24·94	58,730	196	3·42
1944 ..	59,612	20·65	1511	24·72	61,123	186	3·12
1945 ..	61,662	21·14	1840	24·37	63,202	139	2·25
1946 ..	67,247	22·83	1547	22·49	68,794	111	1·65
1947 ..	69,398	23·26	1466	20·69	70,864	130	1·87
1948 ..	67,234	22·26	1326	19·35	68,560	92	1·37
1949 ..	68,812	22·25	1279	18·25	70,091	94	1·37
1950 ..	71,592	22·42	1406	19·26	72,998	80	1·12
1951 ..	72,069	21·98	1291	17·60	73,360	73	1·01
1952 ..	74,196	22·20	1195	15·85	75,391	68	0·92
1953 ..	74,890	22·11	1257	16·51	76,147	48	0·64
1954 ..	73,125	21·33	1207	16·24	74,332	49	0·67
1955 ..	74,407	21·31	1243	16·43	75,650	61	0·82
1956 ..	75,714	21·29	1273	16·54	76,987	62	0·82

While most patients are cooperative and realize the importance of regular ante-natal care, many fail to follow advice given to control early toxæmia because they feel perfectly well; others refuse to enter hospital for treatment when advised to do so. There are still some women who either fail to seek advice at all in pregnancy, or attend for the first time late in pregnancy with severe preeclamptic toxæmia well established.

As has already been stated, only a few of the total number of deaths from preeclamptic toxæmia and eclampsia, as well as from other causes, have been selected for description with relevant details. Appended to each description is a carefully considered comment.

CASE 1.—This patient was a primigravida, aged 22 years, who consulted her doctor when she was about 12 weeks pregnant. Her blood pressure then was 90/70 mm. of mercury, her urine was normal, her weight was 8 st. 10 lb. The hæmoglobin value was 80% of normal, and the blood group O, Rh-positive. The pelvis was radiologically examined, and the "measurements were adequate". At the next consultation, one month later, her blood pressure was 110/70 mm. of mercury and her urine was normal, but she was not weighed.

Six weeks later her blood pressure was 130/80 mm. of mercury, her urine was normal, and again she was not weighed. Subsequent visits were made (to her doctor) at the twenty-sixth and the twenty-eighth weeks; her blood pressure remained at the same level, and her urine was normal; at the twenty-eighth week her weight was 10 st. 11·5 lb.—that is, two stone more than at her first visit 16 weeks before. Advice with regard to diet was given.

At the thirty-second week her blood pressure was 135/80 mm. of mercury, and her urine contained a heavy cloud of albumin. Four days later her urine still contained a heavy cloud of albumin, and there was œdema of the ankles and feet.

The patient was then admitted to hospital—that is, at 33 weeks—and kept under observation for the next six days. No details of treatment are available, but her blood pressure varied between 140/106 and 160/120 mm. of mercury, her urine contained albumin, the quantity varying from "one-sixth" to "one-half", and on one occasion it was "solid" with albumin. Labour was not induced, but began spontaneously six days after her admission to hospital.

It has not been possible to obtain satisfactory information concerning events during the 24 hours preceding delivery, but apparently the patient was moved into a special "eclamptic room". There is no record of actual fits having occurred, but a study of the drugs given suggests that fits may have occurred.

On the sixth day after her admission, labour began spontaneously. During the day she was given various sedatives, including morphine and phenobarbitone and four litres of 25% dextrose solution with calcium gluconate (one ampoule) added to the third bottle.

At 9.30 a.m. on the seventh day after her admission, lower segment Cæsarean section was performed by an honorary obstetrician. A living child was delivered. The birth weight was not recorded. The patient was given a transfusion of two bottles of serum after operation. Catheterization of her bladder did not produce any urine, and she died eight hours after the operation.

TABLE III.

Causes of Death, According to the Sixth Revision of the "International list of Classification of Diseases, Injuries and Deaths".[1]

Cause.	Total Number of Deaths.[2]	Preventible.	Non-Preventible.
Toxæmia:			
Pre-eclamptic toxæmia ..	12	8	4
Eclampsia	19	18	1
Post-partum eclampsia ..	2	1	1
Chronic hypertension	8	5	3
Chronic glomerular nephritis	4	2	2
Hyperemesis	3	2	1
Other toxæmias	5	3	2
Hæmorrhage: ..			
Ante-partum hæmorrhage ..	14	6	8
Post-partum hæmorrhage ..	20	13	7
Puerperal sepsis	17	12	5
Thrombophlebitis and pulmonary embolism	25	6	19
Delivery with complications ..	24	17	7
Abortion	73	54	19
Ectopic gestation	15	8	7
Cerebral hæmorrhage	9	—	9
Other causes of maternal death	12	4	8
Total	262	159	103

[1] Section XI, "Deliveries, complications of pregnancy, childbirth and the puerperium".

[2] Deaths classified as "non-maternal", 57; deaths classified as "maternal", 262; total, 319.

The cause of death was certified as "acute yellow atrophy of the liver and toxæmia of pregnancy". No further information is available, and there was no post-mortem examination.

Comment.—Because of the inadequate records, it is not possible to say whether there was eclampsia or not. There does not seem to be any clinical or other reason to justify the diagnosis of acute yellow atrophy of the liver, the certified cause of death. X-ray pelvimetry at the twelfth week is not in accordance with modern practice, though this did not, of course, contribute to her death. The patient was not weighed between her first visit at 12 weeks of pregnancy and her fourth visit at 28 weeks, an interval of 16 weeks during which she gained 28 lb. The significance of this excessive weight gain does not seem to have been appreciated. She was not admitted to hospital for at least four days after she developed albuminuria. The death was due to toxæmia of pregnancy.

CASE 2.—This patient was a multipara, aged 36 years. She had two children, aged respectively 15 and 12 years. In this, her third pregnancy, she first visited her doctor for ante-natal care at the seventh week, when her blood pressure was 130/80 mm. of mercury; she was not weighed, and her urine was not tested. There is no record of urine examinations during pregnancy until the week before she entered hospital. At the nineteenth week she weighed 9 st. 10·5 lb., and her blood pressure was 110/70 mm. of mercury. At the twenty-third week her blood pressure was 130/70 mm. of mercury, and a further weight gain of 3·5 lb. had occurred.

At twenty-seven weeks she weighed 10 st. 5 lb.—that is, she had gained 14·5 lb. in 16 weeks; moreover, she had gained 5 lb. in the last four weeks. No dietary instructions were given. Three weeks later, at 30 weeks, her blood pressure was 140/90 mm. of mercury and her weight 10 st. 8·5 lb. At 32 weeks her blood pressure had risen to 150/100 mm. of mercury and her weight was 10 st. 9·5 lb. On this occasion, "a faint trace of albumin in the urine" was noted. A salt-free diet and rest in bed at home were ordered, and she was asked to "attend surgery" again in one week. There is no record of the findings at that visit, or of whether she attended.

Nine days after the last recorded visit, she had an eclamptic seizure at home at 2.30 a.m. Her doctor then arranged for her admission to hospital. On her arrival in hospital at 5.30 a.m., she was found to be unconscious, having had another fit in the ambulance. Dextrose solution (20%) was given by intravenous injection; the amount was not stated. At 5.55 a.m. a third fit occurred, and lasted for one minute. Catheterization of her bladder after the fit produced "4 ozs. of urine—solid albumin". Her blood pressure at this time was 160/90 mm. of mercury. The patient did not regain consciousness after this fit.

At 10 a.m., 3 grains of "Luminal" were given intravenously. Penicillin therapy in a dosage of 100,000 units every six hours was started at 12.30 p.m. At 2.20 p.m. she had a fourth eclamptic fit. Paraldehyde (6 dr.) was given, and also dextrose solution by intravenous injection (the strength and quantity were not stated). Classical Cæsarean section was performed by her doctor at 3.20 p.m., and a living child weighing 2 lb. 12 oz. was delivered. After the operation more fluids were given intravenously (the type and quantity were not stated).

A 8 p.m. a fifth eclamptic fit occurred and lasted for eight minutes. Morphine was given (the dosage was not stated), and then a lumbar puncture was performed. The lumbar puncture showed "evidence of cerebral hæmorrhage and cerebral œdema". The patient died 33 hours after delivery. No post-mortem examination was performed.

Comment.—This was a preventible death due to very bad antenatal care. The rise of blood pressure to 150/100 mm. of mercury at 32 weeks, with albuminuria, should have been the signal for immediate admission to hospital for skilled care. Instead the patient was told to go home to rest in bed on a "salt-free" diet, and to return in a week. Before she returned she had two eclamptic fits. This is another case in which a rising systolic and diastolic blood pressure gave warning of trouble ahead, but was ignored.

CASE 3.—This patient was a primigravida, a New Australian, aged 28 years. She spoke very little English. She did not visit her doctor for ante-natal care until "half way through pregnancy". The doctor kept no records of any kind, and therefore, information relating to the ante-natal period is scanty. He stated that the patient was "a big overweight woman", and that "she was well until two or three weeks before her admission to hospital when she had a rise in blood pressure, and some albumin in her urine". She was never weighed. She was given a salt-free diet of restricted carbohydrate content, but the doctor thought that she did not keep to it. Eleven to 18 days later she was advised to go into hospital for treatment for toxæmia, but failed to do so. Four days after that labour began; her doctor found that her urine contained "half albumin", and arranged for her to be taken to hospital by ambulance. "She had convulsions in the ambulance."

On arrival in hospital, the patient was put into the labour ward and given an intramuscular injection of a quarter of a grain of morphine, on the instructions of another doctor who happened to be on the premises at the time. Her own doctor arrived shortly afterwards, and immediately arranged for her transfer to an obstetric teaching hospital.

On examination after the transfer, the patient was found to be comatose, unresponsive to painful stimuli and cyanosed. Tachypnœa was noticed; her pulse was full and bounding, the rate being 106 per minute. Her blood pressure was 220/110 mm. of mercury, and there was œdema of the face, hands and legs. "A specimen of urine boiled solid albumin." The uterus was the size of a 34 weeks' pregnancy, the vertex was presenting in the right occipito-anterior position, and the fœtal heart sounds were audible. There was no dilatation of the cervix, and no uterine contractions could be felt.

Venesection and lumbar puncture were done, but the patient died within eight hours after her admission to hospital. The relatives refused permission for a post-mortem examination, but provided the information that the patient had had œdema of the feet for six weeks and of the hands for four weeks, and headache and epigastric pain for three days.

Comment.—The Committee considered that this was a case of bad ante-natal care by the patient's doctor. At the very least she should have been admitted to hospital under skilled care when he found raised blood pressure and albuminuria about four weeks before the onset of eclampsia. Instead she was given diet instruction and apparently not seen again for at least eleven days. It was noted that the doctor had pleaded "language difficulty" as an excuse for the final outcome; in fact this is no excuse, as the Division of Maternal and Baby Welfare provides printed sheets of questions relating to pregnancy, and also diet instructions, in 17 different languages. This information concerning translation is published in the departmental booklet "Healthy Motherhood".

CASE 4.—This patient was a primigravida, aged 25 years. She first visited her doctor for ante-natal care when she was about six weeks pregnant; her blood pressure was then 118/80 mm. of mercury. The doctor did not keep any records of weight or of urine examinations, but stated that her weight gain was "within normal limits". Her urine remained normal until the last fortnight of her pregnancy. She attended regularly for ante-natal care "monthly to the eighth month and fortnightly to the ninth month", and from the first visit was on a salt-restricted diet. Her blood pressure was normal till the thirty-sixth week, when it was 140/95 mm. of mercury and there was a faint cloud of albumin in her urine. Strict diet instructions were given—that is, reduction of carbohydrate, fat and salt intake. One week later the blood pressure was 190/110 mm. of mercury, and the urine contained "half" albumin. Two days later, at about 38 weeks, the patient was admitted to a private hospital for dilatation of the cervix under general anæsthesia. This operation, together with rupture of the membranes, was performed by the doctor the next day. Some bright bleeding occurred, but no contractions followed. Slight laceration of the perineum occurred at operation. The same day her blood pressure was 210/120 mm. of mercury, and a catheter specimen of urine showed "solid albumin". Castor oil was given at 2 p.m. and followed by an enema at 4 p.m. The patient vomited, but failed to go into labour. No other treatment was given. The next day, the second day after her admission to hospital, she was still nauseated and not progressing, although there was "full drainage". In the early hours of the next morning she had a severe headache, and then had an eclamptic fit. The doctor was notified, and arranged for her to be transferred to an obstetric teaching hospital.

On her admission her condition was "only fair", her blood pressure being 100/60 mm. of mercury and her pulse rate 110 per minute. "The uterus was very tender and there was some slight vaginal bleeding." Her condition rapidly deteriorated in spite of blood transfusions, and the intravenous administration of "Eschatin" and noradrenaline (amounts not stated). She died undelivered three hours after her admission to the teaching hospital.

Post-mortem examination revealed generalized œdema, marked pulmonary œdema, ascites, liver enlargement with a "slight nutmeg appearance", enlargement of the kidneys and a double ureter on the right side; the certified cause of death was "eclampsia".

Comment.—The Committee noted that, although the weight gain was stated to be "within normal limits", no records were kept. This patient should have been admitted to hospital at 36 weeks, when her blood pressure was 140/95 mm. of mercury and her urine contained albumin. Instead, she was not admitted until nine days later, two days after her blood pressure had reached 190/110 mm. of mercury and her urine contained "half" albumin. Cæsarean section performed after the surgical induction of labour had failed might have saved her, although the initial fault was failure by the doctor to realize the significance of the raised blood pressure and albuminuria nine days before her admission to hospital.

CASE 5.—This patient was a multipara, aged 32 years, with a history of three previous confinements. No details were available. In the present pregnancy she was said to have been under the care of her doctor throughout, but no records were made available. She was admitted to hospital one week before term with a twin pregnancy, gross albuminuria, oliguria, headache, blurred vision, nausea and vomiting. On admission she was given one-quarter of a grain of morphine and one one-hundred-and-fiftieth grain of atropine by intramuscular injection. Five hours later she had a fit lasting a few seconds, and she had two more fits 25 minutes and 45 minutes later. She was given a further injection of one-quarter of a grain of morphine, and when she was conscious, a 4 oz. 25% magnesium sulphate solution retention enema was administered. There is no record of the blood pressure during this time. By midday the cervix was one-third dilated, and artificial rupture of the membranes was performed.

At 7 p.m., on catheterization of the patient's bladder, the urine was found to be "almost solid with albumin". Normal delivery of twin girls followed, and the third stage was completed at 9.7 p.m. "Distaquaine Penicillin", in a dosage of 450,000 units twice a day, was ordered, and the first dose was given at 10 p.m.

The patient had eight eclamptic fits between 11.35 p.m. and 2 a.m. the next day, for which she was given one-quarter of a grain of morphine at 11.45 p.m. Six ounces of urine were passed at midnight.

By 2 a.m. her condition was very poor, and venesection was performed; 18 oz. of blood were withdrawn, and intravenous drip administration of 25% dextrose solution was begun. Another one-quarter of a grain of morphine was given at 2.45 a.m. Oxygen was given "when necessary".

At 3.30 a.m. a rectal drip administration of fluid was begun, as the intravenous drip solution was found to be running into the tissues.

By 6 a.m. she was semi-conscious and improving. The next day she was free of fits and was taking fluids by mouth. During the day she passed 24 oz. of urine, containing "seven-eighths" albumin. The blood pressure recordings at 6 p.m. and 9 p.m. were 100/75 and 125/95 mm. of mercury respectively. Her pulse rate varied from 120 to 140 per minute. She was sedated at 9 p.m. with potassium bromide and chloral; catheterization of her bladder at 4 a.m. failed to produce any urine. However, next morning she was "brighter", although still complaining of blurred vision and headache. The urinary output remained very low—never more than six ounces per 24 hours for three days. After this the output improved, but never exceeded 11 ounces until the day before her death, when she passed "large amounts of urine".

The patient developed a cough three days after delivery; she had been receiving penicillin treatment since delivery, and this was continued. In addition, cocillana linctus was given and postural drainage was started. Morphine (one-quarter of a grain) with atropine (one one-hundredth of a grain) was given twice a day, also phenobarbitone (one-quarter of a grain three times per day), and she was on a diet of low protein and low salt content.

Her chest condition failed to improve, and she became gradually more breathless and was found to have some œdema. During the succeeding two days her condition steadily deteriorated. She was very restless on the evening of the seventh day and became unconscious at 8.30 p.m.; she died at 1.10 a.m. on the eighth day of the puerperium.

No autopsy was performed. The cause of death was certified as "acute atrophy of the liver and post-partum toxæmia".

Comment.—The ante-natal care in this case was bad. The doctor kept no records. The patient was not admitted to hospital until one week before term with gross albuminuria, oliguria, headache and blurred vision. She had eclampsia both ante-partum and post-partum.

CASE 6.—This patient was aged 35 years, and had had seven full-time confinements; she had a history of eclampsia with the first and fifth. Of the investigations during pregnancy, only blood-grouping and Rh-typing records were found. There is no prenatal clinic at the public hospital where she attended, and her doctor destroyed her card as she had died. She was admitted to hospital six days before the onset of labour, being considered at that stage very ill, with a blood pressure of 208/130 mm. of mercury, urine containing "half" albumin, and gross œdema of the limbs, trunk and face. Treatment was begun with the intravenous administration of 25% dextrose solution, sedation and bed rest. By the fourth day her blood pressure had dropped to 180/130 mm. of mercury, her urine contained "one-third" albumin, and vomiting occurred. On the fifth day her colour was "dusky", and she was difficult to rouse, but improved on being given oxygen. She was very restless, with slight cough and moist respirations. Labour began at this stage, the period of gestation being 41 weeks, and X-ray examination showing a twin pregnancy.

The patient was so obese that palpation was difficult. One fœtal heart was audible, the other not. Twenty-one hours after the onset of labour Caesarean section was performed under "open ether" anæsthesia, the indications being "no progress, not dilating, pre-eclamptic toxæmia". Further 25% dextrose solution was given intravenously. Living twins were delivered at operation, a male weighing 7 lb. 0·5 oz. and a female weighing 6 lb. 3·5 oz.

Further sedation was given after operation; but eight hours later her pulse was soft and irregular, her colour remained dusky and her respirations moist, and she appeared to be sleeping soundly. One hour later she was sweating, her pulse rate was about 60 per minute, her colour was dusky, and her respirations were moist and stertorous. Her pupils did not react to light, and she gave no reaction to painful stimuli. Her blood pressure was 80/40 mm. of mercury. No more sedation was given; she died two hours later.

In all, over a period of seven days in hospital, she was given in divided doses, a total of 60 ml. of paraldehyde intramuscularly (50 ml. of this being given during the 48 hours before death) and 15 grains of soluble phenobarbitone intramuscularly (12 grains of this in the 48 hours before death), as well as phenobarbitone, half a grain three times a day, and a sedative mixture of potassium bromide and chloral hydrate, half an ounce every four hours; morphine (one-quarter of a grain) was given twice by hypodermic injection during this time.

Comment.—The ante-natal care in this case was totally inadequate, especially in view of the fact that the patient had had eclampsia in two previous pregnancies. Because of destruction of records, it is not possible to determine how long the delay before her admission to hospital had been, after the appearance of serious signs and symptoms. While she was in hospital, excessive doses of sedatives were given.

CASE 7.—This patient was a primigravida, aged 33 years. She first visited her doctor when she was about nine or ten weeks pregnant. There is no record of weight or regular blood pressure readings during the first six months, though it is stated that she was examined regularly every month until the sixth month, and that there were never any abnormalities in the urine. Her blood pressure had been recorded once only, at the twenty-fourth week, when it was 130/80 mm. of mercury, and three weeks later, when it was 130/85 mm. of mercury. Three weeks later still, when her blood pressure was still 130/85 mm. of mercury, she was admitted to the local country hospital, and laparotomy was performed for intestinal obstruction. Numerous adhesions, the result of peritonitis from a ruptured appendix when she was a child, proved to be the cause of the intestinal obstruction. Operation was difficult, and in the course of freeing the bowel of adhesions it was opened in three places, but immediately repaired. A loop of bowel which had been kinked was excised, and lateral anastomosis was performed. The intravenous administration of fluids and gastric suction were instituted after operation. The patient developed "right basal pneumonia, a fæcal fistula, and a burst abdominal wound and thrombosis of the right leg". She improved slowly, but within three weeks again developed symptoms of partial intestinal obstruction.

She was then transferred to an obstetric teaching hospital, where she was treated by the intravenous administration of fluids (4% glucose solution in one-fifth normal saline), and gastric suction; her response was good. She was kept in bed for the rest of her pregnancy and given a salt-free diet because of the development of hypertension and œdema during convalescence. Her blood pressure, recorded daily, varied between 135/90 and 208/140 mm. of mercury; on most days it was 150/100 or 160/100 mm. of mercury. Sedation with chloral (20 grains every three hours) was started. Her urine was tested daily and remained normal.

At 36 weeks, the patient suddenly had an eclamptic fit at 8 a.m. During the fit her blood pressure was 220/140 mm. of mercury; half an hour later it was 170/100 mm. of mercury. Her urine was normal before the fit, but a catheter specimen taken afterwards contained a cloud of

albumin. The patient did not regain consciousness after the fit. Treatment consisted of the administration of oxygen, one-quarter of a grain of morphine, and 8 ml. of paraldehyde by intramuscular injection. Labour was induced at 6 p.m. that day by low rupture of the membranes. Later on the same day the patient died undelivered. The exact time of death is not known, but the fœtal heart was heard 15 minutes before death. Post-mortem Caesarean section was not performed. Post-mortem examination revealed: "Extensive left-sided cerebral hæmorrhage. No evidence of vascular disease. Liver macroscopically and microscopically normal. Kidneys normal apart from congestion and casts in the tubules."

Comment.—This case illustrates the danger of very high and especially quickly rising systolic blood pressure causing cerebral hæmorrhage—which is probably the most frequent cause of death in eclampsia. The urgent use of hypotensive drugs in this case was indicated, and might have saved the patient's life.

Chronic Hypertension Existing before Pregnancy, with or without Superimposed Preeclamptic Toxæmia.

There were eight maternal deaths from this cause, of which five were considered to be preventible. They are included here as a separate group, though no provision is made for them in the International Classification, which includes only maternal deaths from disease "arising during pregnancy".

Two illustrative cases are described.

In addition to the eight maternal deaths mentioned above, of which chronic hypertension was considered to be the primary cause, there were 15 deaths to which it was a contributory factor. In four of these cases the assigned cause of death was cerebral hæmorrhage.

CASE 8.—This patient was a multipara, aged 36 years. Her first pregnancy at the age of 21 years was normal. She had had toxæmia during her second pregnancy (age at the time not stated), and was in hospital for the last three weeks before the delivery. She also had one miscarriage at two months when she was aged 24 years. In this, her fourth pregnancy, she first visited her doctor for ante-natal care at six weeks. Her weight was then 9 st. 2 lb., her urine was normal and her blood pressure was 135/100 mm. of mercury. At the next visit five weeks later, her weight was 9 st. 6 lb., her blood pressure was not recorded and her urine was normal. In the next week she gained 3 lb., and her blood pressure was 140/95 mm. of mercury. No treatment was ordered, and she was not seen again for five weeks. At this visit, at 17 weeks, she weighed 10 st. 2 lb., a total gain of 14 lb. since the first visit 11 weeks before; her blood pressure was 160/100 mm. of mercury, but her urine remained normal. She was then put on a diet of low salt content and examined again in one week. By this time she had gained a further 2 lb. in weight, but her blood pressure was 125/85 mm. of mercury. At the next visit two weeks later at the twentieth week—her weight was 10 st. 8 lb., another gain of 4 lb., her blood pressure was 150/100 mm. of mercury and her urine was normal. During the next seven days her weight dropped by 5 lb., but her blood pressure rose to 170/100 mm. of mercury; one week after this it was 146/96 mm. of mercury, and her weight was 10 st. 5 lb. Two weeks later, at 25 weeks' pregnancy, her weight was 10 st. 11 lb., her blood pressure was 146/100 mm. of mercury and her urine was normal. Five days later her weight had dropped again to 10 st. 8 lb. At about 28 weeks' pregnancy her blood pressure was 160/104 mm. of mercury. At 29 weeks she weighed 11 st. and her blood pressure was 170/110 mm. of mercury; she was admitted to hospital the next day.

In hospital, her urine was found to be normal, there was no œdema, and the blood urea content was 38 mg. per 100 ml. The patient was allowed to go home 10 days later with a blood pressure of 130/90 mm. of mercury. Three days later the weight recorded was 11 st. 3 lb., and in another 14 days (at 33 weeks' pregnancy) it was 11 st. 9 lb. Her blood pressure was not recorded on these two occasions, but her urine contained "one tenth" albumin. Two weeks later, at 35 weeks, her blood pressure was 180/120 mm. of mercury, her urine contained "one third" albumin and there was obvious ankle œdema. Only then was the patient readmitted to hospital.

On that day her blood pressure rose to 200/120 mm. of mercury. Treatment was as follows: 5 ml. of paraldehyde were given by intramuscular injection and also 10 ml. of 50% magnesium sulphate solution intramuscularly every four hours, and 500 ml. of 25% dextrose solution were given intravenously every eight hours.

The next day the patient developed a temperature of 101·4° F. due to cellulitis of the buttock. On the evening of the same day the membranes ruptured spontaneously, labour began two hours later, and normal delivery followed in seven and a half hours. After delivery the blood pressure fell to "normal", but the cellulitis of the buttock failed to retrogress; 100,000 units of penicillin were given every six hours for one week after the confinement. "Trisulfa" was also given (dosage not stated). Culture of a vaginal swab gave negative results. Examination of a catheter specimen of urine revealed many red cells and occasional leucocytes, but attempted culture produced no growth of pathogenic organisms.

One week after delivery the patient was still pyrexial in spite of treatment. The temperature varied from 98° to 102° F. "Aureomycin", in a dose of 500 mg. every six hours, was given instead of penicillin.

Five days later an abscess of the buttock was incised, and the pus obtained was sent for culture. After this her temperature and blood pressure were stated to be "normal". Two days later—14 days after delivery—the patient suddenly became breathless and died within five minutes.

There is no record of any post-mortem examination having been made. The cause of death as stated on the certificate is: "Pulmonary embolism; uterogestation—delivered; pre-eclamptic toxæmia of pregnancy (puerperal)".

Comment.—This was evidently a case of chronic hypertension with superimposed preeclamptic toxæmia of severe degree. Little or no notice seems to have been taken of the consistently abnormal weight increase found at successive ante-natal visits. At the thirty-third week of pregnancy the urine contained albumin, but the patient was not admitted to hospital till two weeks later, when her blood pressure was 180/120 mm. of mercury and her urine contained "one-third" albumin. The cellulitis of the buttock was probably due to the repeated (four-hourly) intramuscular injection of magnesium sulphate solution. This is recognized treatment for eclampsia, but is not usually given in preeclampsia. The 5 ml. injections of paraldehyde every four hours no doubt were an additional irritant and possible source of infection. The Committee considers that the ante-natal care was inefficient and that the patient should have been admitted to hospital much sooner.

CASE 9.—This patient was a multipara, aged 35 years, who was known to have had three pregnancies complicated by "toxæmia". One child was born prematurely, "two were stillborn at seven months". There was "albuminuria" in each pregnancy. Ante-natal care in this, her fourth, pregnancy was given by the patient's own doctor. The only information given by him concerning this pregnancy was as follows: "No abnormal gain in weight. Blood pressure not over 135 systolic during pregnancy, the diastolic pressure was not abnormally raised. Albumin was present during the last month about five per cent. On rest and salt free diet."

Ten weeks before the expected date of delivery, the patient had an ante-partum hæmorrhage, and was delivered at home two hours later of a stillborn premature child. "The patient passed urine at the time of delivery and a small amount the following day—none since."

Five days after delivery she was admitted to an obstetrics teaching hospital with anuria and a blood pressure of 220/130 mm. of mercury. Pathological investigation revealed that the hæmoglobin value was 5·5 grammes per 100 ml., and "the blood urea was greatly raised". Treatment consisted of one blood transfusion, an intragastric drip administration of peanut oil and glucose emulsion (1 litre in every 24 hours), and intravenous administration of dextrose solution (10%) "when necessary". The fluid intake and urine output were charted daily. In spite of treatment, the patient died on the eighteenth day of the puerperium, 13 days after her admission to hospital.

The post-mortem findings were: "acute pulmonary œdema; bilateral renal cortical necrosis; left ventricular hypertrophy."

Comment.—This was a case of chronic hypertension complicating the fourth pregnancy. There was a bad obstetric history, including albuminuria in each pregnancy, and two stillborn babies at seven months. In this pregnancy albuminuria was present from about the twenty-fifth week, yet the patient was not admitted to hospital. Ante-partum hæmorrhage, probably accidental, occurred at

the thirtieth week, followed by birth of a dead baby. In spite of anuria the patient was allowed to remain for five days at home after delivery before being transferred to an obstetric teaching hospital. There is much to be said for the immediate transfer of all patients with obstetric anuria to a hospital where there is a special kidney unit, including an artificial kidney.

CASE 10.—This patient was a multipara, aged 41 years. She had had nine previous full-time confinements (details not known), and was known to have chronic nephritis and hypertension, which had complicated the previous pregnancies. She had also had one miscarriage. No record of the examinations during pregnancy was kept by the doctor; therefore the available information is inadequate. The patient did not attend regularly for ante-natal supervision, and the doctor records this as "unsatisfactory".

It is not known at what stage of pregnancy the patient was first examined by the doctor. No records of weight, urine examination or blood pressure readings were kept. However, the doctor stated, that "urine examination was irregular and the urine was always loaded with albumin", and that the blood pressure taken "on many occasions was always in the range of 250/130". Termination of pregnancy was advised, but the patient refused; she also refused to go into hospital for treatment until she was seven months pregnant.

On her admission to hospital at this stage (seven months), she had massive albuminuria, hypertension and œdema of the feet and ankles; she also had a slight "show"; the fœtal heart was audible. Bed rest and a diet of low salt content were maintained for only five days. During this time a slight constant loss of dark blood per vaginam was evident. On the sixth day the patient was allowed to get up and move about because the blood loss had stopped. Five days later she was allowed to go home.

No further treatment had been ordered, no second opinion was requested, and the advisability of transferring the patient to a specialist unit was apparently not considered by the doctor. No investigations with regard to blood ureal level, hæmoglobin value, etc., were made.

Nine days later the patient was readmitted to hospital at 4.45 p.m., with a further vaginal hæmorrhage. On her admission, the loss was found to be "only slight", but her skin was noted to be "cold and clammy" and her colour was congested. Half an hour later she complained of epigastric pain and vomited. At 6 p.m. she developed paralysis of the left arm, slurred speech and twitching of the face. At 11.15 p.m. a quarter of a grain of morphine was ordered and given. This is the only information available during this period.

A stillborn infant was delivered at 10.30 a.m. the next day. By 3.30 p.m. the patient's condition was "much worse"; her temperature was 105° F. and her pulse rate 140 per minute. On the day after delivery, her temperature was 102° F. at 2 p.m. No further details of her condition are available. She died at 1.50 a.m. the next morning. No post-mortem examination was performed.

Comment.—This was a case of malignant chronic hypertension. It would have been better to terminate the pregnancy as early as possible, but the patient refused it. She should not have been allowed to go home after the first admission to hospital at the seventh month of pregnancy. The immediate cause of death was obviously cerebral hæmorrhage.

Other Toxæmias.

There were eight deaths from "other toxæmias". These include hyperemesis gravidarum (three deaths), liver atrophy, hepatitis, "combined renal and liver failure" and systemic lupus erythematosus.

CASE 11.—This patient was aged 29 years. She had had one child two years previously, and had suffered from hyperemesis throughout that pregnancy. In this, her second pregnancy, she first visited her doctor for ante-natal care when six weeks pregnant; thereafter she was examined "at least three times weekly either at surgery or at her home" on account of nausea and vomiting which she had had from the beginning of the pregnancy. Her blood pressure, recorded monthly for three months, was always within normal limits—125/74 to 130/70 mm. of mercury. Her urine did not contain albumin until she was 11 weeks pregnant. She was weighed on two occasions, at the first visit when she weighed 9 st. 7 lb., and one month later when she was stated to weigh 10 st.—that is, a weight gain of 7 lb. There is no record of the treatment given to control the nausea and vomiting.

At about 11 weeks of pregnancy, she was admitted to a private hospital for observation and treatment of the hyperemesis. Her doctor advised termination of pregnancy (reason not stated), but the patient refused, saying that she would like to have this baby and then be sterilized. The matron of the private hospital to which she was admitted said that she "was a tall frail woman unusually disinterested in food"—taking only small quantities which had to be spoon fed to her. She took very little fluid. Extracts from the hospital reports reveal that her urine persistently contained acetone, often in large amounts. Vomiting occurred on most days and was sometimes excessive. On the day after her admission to hospital, 3000 ml. of dextrose-saline solution were given. No further treatment was ordered.

On the fourteenth day after her admission to hospital, she vomited several times; she was given a "Dexsal" draught, and her doctor said she could go home. However, as she vomited frequently the next day and her urine contained "heavy acetone", she was kept in hospital. A further 3000 ml. of dextrose-saline solution were given next day. For the following six days, marked acetonuria continued. On the sixth day—22 days after her admission to hospital—she vomited at 3 a.m. and her urine contained "heavy acetone"; but the doctor allowed her to go home.

Two days later she was visited at home by her doctor, who reported that she said she felt better and was eating more. However, one day later her husband sent for the doctor because "her condition was deteriorating and very little urine had been passed, although the patient had eaten well". Two hours later she was admitted to a different private hospital, and she died within one hour. The cause of death as stated on the certificate was as follows: "Acute cortical necrosis, toxæmia of pregnancy, hyperemesis gravidarum". No post-mortem examination was made.

Comment.—This death was regarded as preventible, and due to faulty management. Records of the patient's progress in hospital are largely lacking, as her chart had been lost. Thus there are no adequate records of pulse or temperature or of fluid intake or urine output, nor did the urine seem to have been tested for bile. Fluid replacement was insufficient. She was discharged from hospital though still vomiting, and while the urine contained "heavy acetone". There was no post-mortem examination, and no real evidence of renal cortical necrosis. In the Committee's opinion, death was due to hyperemesis gravidarum.

Hæmorrhage.

Ante-Partum Hæmorrhage.

Deaths due to placenta prævia and other causes of ante-partum hæmorrhage during the years 1950 to 1956 totalled 14; nine were due to placenta prævia, and of these, four were considered to be preventible.

Death mostly occurred in these cases of placenta prævia either because blood transfusion was not given, or because those in charge of the case failed to recognize that when hæmorrhage from placenta prævia is of such severity as to require a transfusion, immediate active treatment, involving termination of the pregnancy by Cæsarean section or other method as circumstances indicate, is imperative.

CASE 12.—This patient was aged 41 years. She had had three previous pregnancies, the last ten years earlier. All the data, which were exceedingly scanty, had to be obtained by the supervisory nurse from the hospital records. No information could be obtained from the patient's doctor in spite of four letters and two telephone calls. He said that he kept no records and remembered nothing about the case.

In this pregnancy the patient had been admitted to hospital on two occasions, about the thirty-fourth and thirty-sixth weeks, with "hypertension and slight P.V. loss and suspected placenta prævia". She was discharged after six days on the first occasion, and after seven days on the second. She was again admitted about the thirty-eighth week, and 13 days afterwards surgical induction of labour was performed; but neither the reason for her admission to hospital nor that for the induction of labour is stated. The presentation was "shoulder rotated to breech". A live child weighing 7 lb. 12 oz. was born 12 hours later. Fatal intrapartum and post-partum hæmorrhage occurred. The only treatment mentioned was the administration of "Pitocin" and ergometrine.

Comment.—Because of the dearth of information, it can only be surmised that the ante-partum hæmorrhage for which the patient was admitted to hospital on two occasions was due to placenta prævia, and that this was also the reason for her readmission at the thirty-eighth week for the surgical induction, and the cause of the fatal intrapartum and post-partum hæmorrhage. There is no indication that the seriousness of recurrent ante-partum hæmorrhage in the later weeks of pregnancy was appreciated at any time, or that the treatment of the post-partum hæmorrhage was adequate. No blood transfusion was given. Though the hospital was a small one in a country town, it was only 30 miles from a large centre where stocks of blood are always available, and in the town itself a blood-donor panel had been organized.

CASE 13.—The patient was a multipara, aged 26 years. The doctor who undertook her ante-natal care had managed her two previous normal confinements. According to him, she was a good mother and came from a well-kept home. She attended regularly for ante-natal care; details are not available, but apparently she progressed normally up to the thirty-eighth week of pregnancy. At this stage she suddenly had a massive ante-partum hæmorrhage. A diagnosis of placenta prævia was made, and she was transferred by ambulance to the nearest country hospital some miles away.

On her arrival at the hospital, her condition was poor, but bleeding had ceased. Abdominal examination revealed the vertex presenting in the left occipito-anterior position, but not engaged; the fœtal heart sounds were audible. She was given an injection of morphine (dosage not stated), and blood was taken for grouping and hæmoglobin estimation. Her pulse rate, taken half-hourly, varied from 90 to 96 per minute all that day, and vaginal bleeding recommenced—a small amount only. During that night she had a fairly heavy loss, but her pulse rate remained satisfactory—80 to 88 per minute—and the pulse was of good volume. The next day she had a slight vaginal loss of dark blood, the blood group was found to be B, Rh-positive, and blood was ordered from Sydney. The hæmoglobin value was 7·2 grammes per 100 ml. No hæmorrhage occurred during that night, and only a slight one during the next 24 hours. Three days after the patient's admission to hospital, and two days after it had been ordered, the blood arrived. Two bottles were then given, but no other active treatment was undertaken, although she continued to have persistent vaginal loss of blood throughout this day and the subsequent three days.

On the evening of the seventh day, another transfusion of blood was begun, but after 100 ml. had been given the patient had a reaction—nausea, headache, pain in the renal areas, and a rigor—so the transfusion was stopped. One sixth of grain of morphine was given. Her temperature was 102° F. and her pulse rate 112 per minute.

The patient had a slight hæmorrhage at 3 a.m. the next day and passed a small clot; then at 3.45 a.m. she had a severe hæmorrhage, and became very shocked. The loss was estimated at two pints. The blood pressure was recorded as "55/?". The honorary medical officer on call was notified, and a quarter of a grain of morphine was ordered and given.

The resident medical officer gave 30 mg. of "Methedrine" intravenously, and then a "cut-down" transfusion of one bottle of blood followed by two bottles of serum. From 3.30 a.m., 15 minutes before the last severe hæmorrhage, uterine contractions had been noted. The blood loss per vaginam, though less after transfusion, had continued. At 6 a.m. it again became excessive, the fœtal heart could not be heard and the patient's condition steadily deteriorated. A further "cut down" was done on the left ankle, and two bottles of "Dextran" were given, followed by another bottle of blood. The doctor in charge of the patient decided at 6.30 a.m. "to improve her condition by transfusion and then do a Cæsarean section"; but by 8.30 a.m. the patient was pulseless. Nothing further had been done except to give oxygen. She finally collapsed and died at 10.30 a.m., eight days after her admission to hospital. No post-mortem examination was performed.

Comment.—The Committee considered that the doctor had acted correctly in arranging for the patient's immediate admission to hospital. However, the management within the hospital was at fault. The patient, who had had a massive hæmorrhage before her admission, was treated by blood transfusion only. When hæmorrhage in placenta prævia is of such severity as to require transfusion, immediate active treatment by Cæsarean section or other means is indicated. In this case particularly there was no reason for delay, as the pregnancy had reached 38 weeks, and the baby was therefore viable.

CASE 14.—This patient was a multipara, aged 29 years. The details of her previous obstetric history are unknown, apart from the fact that she had three children, and had never had a miscarriage. She visited her doctor for the first time, in this her fourth pregnancy, at approximately the fourth month, because of vaginal bleeding, which had been persistent for two days. Her doctor sent her to an obstetric hospital, where she was admitted next day. There she gave a history of vaginal bleeding for four days, and "passage of a ? fœtus two days ago". She was found to be very obese. Her pulse rate was 90 per minute, her blood pressure was 130/100 mm. of mercury, and she was bleeding slightly per vaginam. There was a soft abdominal mass (thought possibly to be the uterus) rising out of the pelvis and reaching to the umbilicus. There was no abdominal tenderness or rigidity. The cervix was closed. The breasts were "secreting colostrum". A diagnosis of threatened miscarriage or ante-partum hæmorrhage was made. The patient's hæmoglobin value was 11·1 grammes per 100 ml., and her blood group was A, Rh-positive. Next day her blood pressure was 125/85 mm. of mercury, and slight vaginal bleeding occurred, lasting for half an hour. On the following day bleeding had apparently ceased. She was then discharged from hospital and told to return to the out-patient department in two weeks. "Polyhæmin" was ordered.

Eight days after her discharge she was referred back to the hospital by the doctor with a note stating: "Since discharge she has been bleeding. I feel that she may need a curette." It was stated that, on examination of the patient in the out-patient department, there was "no evidence of bleeding. Fundus up to umbilicus. Cx. soft and not dilated". X-ray examination revealed a fœtal skull "of about twenty-two weeks". These findings were confirmed by the resident medical officer on duty, who did not consider that there was any indication for her readmission to hospital. He requested her to return to the out-patient department five days later, and she was given an appointment.

On the next day, however, the patient's own doctor admitted her to a private hospital, where he curetted her uterus at 10.30 a.m. the same day. At the curettage "she had about fifteen ounces blood clot and macerated fœtus of four months gestation". She collapsed and died at 7.25 p.m. that evening. The cause of death as stated on the certificate was: "Pulmonary embolism: debility."

Comment.—This case seems to have been completely mismanaged, both by the public hospital and by the patient's own doctor. The patient was discharged from hospital while hæmorrhage was occurring, only three days after her admission at the request of her own doctor for threatened miscarriage; eight days later she was sent back by her doctor because of continued vaginal bleeding, but was refused admission. On the next day she was admitted to a private hospital by her own doctor, and early on the same day he curetted her uterus apparently without any observation or preparation, and in spite of the absence of any obvious urgency. It is well known that removal of a four or five months' fœtus by curettage, especially when the cervix is closed, can be an extremely difficult, shocking and hazardous procedure. The death, which occurred a few hours later, was no doubt due to shock and hæmorrhage. Though pulmonary embolism was certified as the cause of death, there was no real evidence of it, and no post-mortem examination.

Post-Partum Hæmorrhage.

The histories of 20 fatal cases of post-partum hæmorrhage studied by the Committee indicated that the majority of deaths were preventible. In most of these cases it was found that the chief avoidable factor was not lack of facilities, but unsatisfactory management of the arrest of the hæmorrhage with inadequate "replacement" therapy. The blood transfusions given were, in a number of instances, too little and too late.

CASE 15.—The patient was aged 29 years. Her home conditions were bad. She had had seven previous confinements, including one forceps delivery, two breech deliveries and one Cæsarean section (the indication for which is not known). After delivery of her last child she had a very severe post-partum hæmorrhage requiring blood transfusion. In this, her eighth pregnancy, she first consulted her doctor at eight weeks. At this time her general health was said to be "very bad". She was debilitated

and anæmic, and the hæmoglobin value was only 7 grammes per 100 ml. A blood transfusion was given, and thereafter the hæmoglobin value was 14·5 grammes per 100 ml. Iron tablets were ordered for the remainder of the pregnancy.

Ante-natal supervision was irregular, owing to lack of cooperation by the patient. Her weight rose from 7 st. 4·5 lb. at 12 weeks to 9 st. 8lb. at 32 weeks. Her blood pressure rose from 130/70 mm. of mercury at eight weeks to 150/80 mm. of mercury at 20 weeks, 160/90 mm. of mercury at 28 weeks, and 168/105 mm. of mercury at 34 weeks. Her urine was free of albumin until the thirty-fourth week. During the pregnancy her doctor tried on several occasions to persuade her to enter hospital for treatment, but she refused, on the grounds that she "had no one look after the children".

She was admitted to hospital in labour at approximately 36 weeks' pregnancy, when examination of her urine showed it to contain "$\frac{1}{16}$" to "$\frac{1}{8}$" albumin on the first two days and none after that. Her blood pressure varied between 168/105 and 170/110 mm. of mercury.

On her admission to hospital, the vertex was presenting in the left occipito-anterior position, and the fœtal heart rate was 140 per minute and regular. The patient gave a history of recent influenza and had right lower thoracic pain on respiration. Chest examination revealed no abnormality. An abdominal X-ray examination suggested the possibility of placenta prævia; but clinical examination revealed a well-engaged head, and there was very little vaginal bleeding during labour. In view of these findings and of the patient's respiratory history, it was decided that Cæsarean section should be avoided. The possibility of uterine rupture thorugh the old Cæsarean scar was borne in mind, and the nursing staff was warned to report changes in the patient's condition.

Labour progressed slowly, and the membranes ruptured at 3 p.m. on the following day. Forceps delivery under ether anæsthesia for maternal and fœtal distress was performed at 2.45 a.m. next morning. The "maternal distress" in this instance was the high blood pressure of 160/110 mm. of mercury, poor contractions and albuminuria. A small intrapartum hæmorrhage of 2 oz. occurred early in labour, but stopped spontaneously. However, after delivery, the patient had an excessive hæmorrhage. During 25 minutes she lost about 88 oz. of blood. Ergometrine (0·25 mg.) was not given until 22 minutes after delivery, about three minutes before manual removal of the placenta for "uncontrolled hæmorrhage" was undertaken. Manual removal proved difficult, as "there was no uterine wall palpable over the placenta". A check after the manual removal revealed "an intact thin membrane" at the placental site.

"Pitocin" (1 ml.) was given on completion of the manual removal. After this the hæmorrhage ceased. Three-quarters of a bottle of serum was given five minutes after completion of the third stage, and followed by three-quarters of a bottle of blood 15 minutes later; the transfusion did not run well because of poor veins due to transfusions during previous labours. The patient's condition deteriorated rapidly, and she died three-quarters of an hour after the manual removal.

A post-mortem examination was performed, and the findings were as follows: "A small old hæmoperitoneum with sticky black altered blood over the liver." A uterine wall hernia was discovered. There was no evidence of rupture, but there was a small area of blackened uterine wall at the periphery of the hernia, which may have been a source of hæmorrhage.

Comment.—This patient had a past history of Cæsarean section. Manual removal was difficult, because the placenta was attached to the paper-thin Cæsarean section scar. The patient lost over four pints of blood in 25 minutes during the third stage, before ergometrine was given three minutes before manual removal. The Committee considered, in view of her past history, that this patient should have been under the care of a consultant. Management of the hæmorrhage was poor, and blood transfusion was inadequate, only three-quarters of a bottle of blood being given.

CASE 16.—This patient was a primigravida, aged 33 years. She first attended her doctor when she was approximately four months pregnant. On this occasion her blood pressure was 130/70 mm. of mercury, her urine was normal, and her weight was 11 st. Dietary advice was given.

The next visit was made one month later; her blood pressure was then 112/64 mm. of mercury and her urine was normal, but she had gained 9 lb. in weight and had œdema of the hands, face and legs. She was put on a salt-free diet of low carbohydrate content, and advised to take 0·5 dr. of magnesium sulphate per day. Two weeks later she had gained another 2 lb., but the œdema was "much less".

The next three visits were made at fortnightly intervals, and her weight gain totalled 6 lb. during this time. Her blood pressure never exceeded 120/80 mm. of mercury, and her urine was always normal. At the thirty-fourth week the vertex was presenting in the left occipito-anterior position, but was not engaged. The fœtal heart sounds were heard.

At 36 weeks, her blood pressure was 130/90 mm. of mercury. The patient then had acute bronchitis, and was treated at home for approximately three weeks.

At the last visit, made about one week before term, her blood pressure was 156/100 mm. of mercury, her urine contained "one-third" albumin, and she had œdema of the face and feet. The patient was then admitted to hospital. Treatment consisted of rest in bed, a salt-free diet of low carbohydrate content, magnesium sulphate every morning and pentabarbitone, 3 grains every night. Albuminuria continued, the albumin content of the urine varying from a "slight cloud" to a "heavy cloud".

On the sixth day, phenobarbitone, 0·5 grain three times a day, was ordered. In spite of full treatment, albuminuria continued, and her blood pressure varied between 136/80 and 150/90 mm. of mercury.

Medical induction of labour with stilbœstrol, two tablets every half-hour for five doses, castor oil and an enema, was tried, and proved unsuccessful at first. A second attempt on the seventh day, "Pitocin" (two doses of 3 min.) being substituted for the stilbœstrol, produced good contractions—which later ceased.

Surgical induction of labour was performed on the eleventh day after the patient's admission to hospital, and was followed by nine doses of "Pitocin" (3 min.) at approximately hourly intervals. At the end of this treatment "the head was still high". The patient was having irregular mild contractions only. At 3 p.m. on the twelfth day, 29·5 hours after the surgical induction, lower segment Cæsarean section was performed. A living child, weighing 9 lb. 4 oz., was delivered. Transfusion of stored blood was started in the operating theatre (as a routine), and was still in progress when the patient returned to the ward. Three hours later she had a massive vaginal hæmorrhage. This was treated at first with ergometrine (1 ml. by intramuscular injection) and fundal massage. The transfusion of blood (second bottle) was still running. "Coramine" (2 ml.) was given 10 minutes later. Hot douching was tried, and then at 7.30 p.m., 20 minutes after the first dose, a further dose of 1 ml. of ergometrine was given, intravenously this time, and also 1 ml. of "Pitocin". In spite of these measures, the hæmorrhage continued, and the patient collapsed and died half an hour later.

In all, the patient received 61 min. of "Pitocin", and only one and a half pints of stored blood, plus a little "saline", the administration of which was started just before she died.

Comment.—The high head in a primigravida at term should have suggested the possibility of disproportion, especially when the head continued high after medical and surgical induction of labour. "Pitocin" in such a case is dangerous and artificial rupture of the membranes undesirable. Better treatment would have been the performance of lower segment Cæsarean section without any previous attempts at induction of labour, when the toxæmia failed to respond to conservative management. In the absence of a post-mortem examination, no reason is apparent for the massive post-partum vaginal hæmorrhage after the Cæsarean section.

CASE 17.—This patient was aged 39 years. She had had five miscarriages, one stillbirth and two full-time confinements. There was a history of difficult labour and a forceps delivery in one of the full-time confinements, and a post-partum hæmorrhage after another. Two years before the present pregnancy she had suffered from "toxic post-influenzal myocarditis", and had then been advised against further pregnancies. In this, her ninth pregnancy, the date of the last menstrual period was not known, as she did not attend her doctor until approximately 10 weeks before she thought the baby was due. At the first visit she was found to be anæmic (her hæmoglobin value was 11 grammes per 100 ml.) and was treated with liver and iron (dosage not stated); blood grouping was not

done. Her blood pressure was 120/85 mm. of mercury. Her subsequent attendances were irregular; the patient brought a specimen of urine with her on only two occasions, on both of which it was normal. The doctor stated that she "did not weigh herself", nor did he weigh her as a routine measure. Her blood pressure varied from 120/85 to 135/85 mm. of mercury.

She was admitted to hospital "near term" with a blood-stained vaginal discharge. Her urine was normal; she was not in labour. The next day a medical induction of labour was tried—three doses of "Pitocin" (5 min.) were given (the time of injection is not stated). Labour began the same afternoon; the membranes ruptured seven and a half hours later. Twenty-five minutes later a living infant, weighing 6 lb. 13 oz., was delivered by low application of forceps, "as the head did not progress, although on the perineum, and the patient was uncooperative".

One hour later the placenta was still retained, there was "moderate bleeding" and manual removal was performed after "pressure on the abdomen" had failed. Further hæmorrhage occurred after manual removal of the placenta. "Pitocin" (1 ml.) and ergometrine (1 ml.) were given, and an intravenous infusion of "glucose saline" was started; but no blood was given, and the patient collapsed and died less than 15 minutes after the manual removal.

Comment.—This death from post-partum hæmorrhage was preventible and due to bad management. There is no indication that any of the well-tried methods for the control of post-partum hæmorrhage were used, such as fundal massage or bimanual compression of the uterus, etc. No blood transfusion was given.

CASE 18.—This patient was aged 41 years. She had had two miscarriages and two normal confinements at term. In this pregnancy she had regular supervision by her doctor, and no abnormality was found at any time. At the fifth month the hæmoglobin value was 12·5 grammes per 100 ml. Labour began at term, and one and a half hours later she delivered herself of a baby weighing 7 lb. 8 oz. Third-stage hæmorrhage occurred, the loss being estimated at 40 oz. in all, and was apparently treated by the administration of "Pitocin" (1 ml.), ergometrine (1 ml.) and "Methergin" (1 ml.); the routes of administration were not stated. After one hour, manual removal of an "adherent" placenta was done. Immediately afterwards the patient appeared to be shocked; warmth was applied, and quarter of a grain of morphine, oxygen by the nasal route and "Coramine" (1 ml.) were given. Twenty minutes later a blood transfusion was set up, but the patient died before it started.

Comment.—The cause of death was post-partum hæmorrhage. The method of control is stated as "manual removal of adherent placenta"; but no mention is made of fundal control, or of the use of any other method of arresting hæmorrhage except the administration of "Pitocin", ergometrine and "Methergin". The Committee considered that blood transfusion should have been given before manual removal was attempted.

CASE 19.—This patient was a primigravida, aged 18 years. Ante-natal supervision by her doctor was regular from the third month. Her blood group was O, Rh-positive, and her hæmoglobin value 69%. Her blood pressure was stated to be "always below 120/80", but there was "difficulty with weight control", and because of this, she was admitted to a private hospital at the thirty-seventh week. On her admission, the blood pressure was found to be 166/100 mm. of mercury and her urine normal. Labour began spontaneously on the day of admission, and the baby, weighing 8 lb., was delivered by low application of forceps about four hours afterwards, the head having been on the perineum for one hour. The placenta, to which a large blood clot was adherent, was born naturally, the entire labour lasting four hours. A perineal tear (degree not stated) was repaired immediately after the birth of the placenta with catgut, deep and superficial sutures being used; but whether this was done under general or local anæsthesia is not stated. "Excessive" post-partum hæmorrhage began apparently just after the perineal repair was completed. Attempts to control it by "external massage and control of fundus", in addition to the administration of "Pitocin" and ergometrine, were unsuccessful. Though blood had been requested from the Red Cross Blood Transfusion Service, there was, for some reason not quite clear, considerable delay in its arrival, so that only one litre was given; the patient died 10 minutes after the second litre was started.

Comment.—The cause of death was post-partum hæmorrhage. Blood transfusion was again too little and too late.

CASE 20.—This patient, aged 37, years had had six children and one miscarriage. In this, her eighth pregnancy, she first consulted her doctor when she was 20 weeks pregnant. Her weight at this time was 11 st., and her blood pressure 140/88 mm. of mercury.

Progress was normal till 36 weeks, when she had a heavy fall on her abdomen; she was admitted to hospital five days later with premature rupture of the membranes.

Two days later, as she was not in labour, simple medical induction was attempted. She was receiving 1,000,000 units of penicillin daily (presumably as a prophylactic measure), but it is not known on which day this treatment was begun. Castor oil and a soap and water enema failed to induce labour. There is no record of any pyrexia during this time. Five days later a second medical induction, including the half-hourly administration of 3 min. of "Pitocin" for five doses, was started. Labour began half an hour after completion of this induction, and 40 minutes later a premature infant was delivered normally.

"Excessive hæmorrhage occurred during delivery and following the birth of the child." Ergometrine and "Pitocin" were given (in amounts not stated), and bi-manual compression was done. After 20 minutes an attempt was made to express the placenta by pressure on the abdomen. Bleeding continued to be excessive, and finally manual removal was undertaken. By this time the patient was in a shocked condition; therefore, an attempt was made to give fluids intravenously while the manual removal was in progress. The veins were very collapsed; only 100 ml. of serum were actually given. Manual removal was unsuccessful "as the placenta was found to be adherent, and failed to come away even in pieces".

A decision to perform hysterectomy was then made; two hours and 40 minutes after delivery of the child, the patient was transformed in a collapsed condition to the district hospital about one mile away. She was given oxygen continuously on her arrival, but died in five minutes. A post-mortem was performed by the doctor in charge of the case. He verified the presence of "placenta accreta".

Comment.—The cause of death was adherent placenta and post-partum hæmorrhage. The Committee considered that in this case too little fluid was given intravenously—100 ml. of serum. It should have been possible to cut down on the collapsed veins, but no attempt was made to do so. Blood should have been given, and the patient adequately resuscitated before she was transferred by ambulance to another hospital. Every hospital accepting women for confinement should have either blood available, or an organization for obtaining blood and an emergency blood-giving team.

Puerperal Sepsis.

Altogether 17 cases of maternal death attributed to puerperal sepsis have been investigated; of these deaths 12 were considered to be preventible.

In many of these cases a wide range of antibiotics was given, although in some cases no culture or sensitivity tests had been done.

Only one death followed an entirely normal delivery. Five deaths occurred after early surgical rupture of the membranes.

CASE 21.—This patient was aged 31 years and was pregnant for the second time. During her first pregnancy the previous year year she had developed toxæmia, for which induction of labour was attempted. This failed, and a lower segment Cæsarean section was performed. She had mild pyrexia in the puerperium. During this, her second pregnancy, she attended the out-patient department of an obstetric teaching hospital. She was first examined when she was two months pregnant. Her blood group was A, Rh-negative; the X-ray findings in the chest, and the hæmoglobin value were normal and the Wassermann reaction was negative; her blood pressure was 120/70 mm. of mercury, her urine was normal and her weight was 13 st. 12 lb.

One month later her blood pressure was 140/70 mm. of mercury, her urine was normal and she had lost 3 lb. in weight. At four months her blood pressure was the same, her urine was normal, and she had lost another 1 lb. in weight.

Three days later her weight had increased by 4 lb. One month later she had gained 5 lb., and her blood pressure was 155/70 mm. of mercury. Two weeks after

this, her blood pressure was 170/70 mm. of mercury and her weight had increased by a further 6 lb. The patient was by this time seven months pregnant, and her weight was 14 st. 9 lb. Her urine had remained normal throughout.

During the next two months her weight rose to 15 st. 7 lb., and her blood pressure varied from 150/70 to 170/90 mm. of mercury.

At 35·5 weeks, her blood pressure was 170/100 mm. of mercury, and she was admitted to hospital for rest. Ten days later she was allowed to leave hospital, because her her blood pressure had settled to 120/80 mm. of mercury.

Within one week her blood pressure had again risen to 170/110 mm. of mercury. Her weight was 15 st. 12 lb. and her urine was normal. She was then readmitted to hospital.

On the following day, and at two-day intervals during the next 11 days, vaginal examinations were made, with finger dilatation of the cervix—a total of seven vaginal examinations.

On the eleventh day "intermittent 'Pitocin' injections" (dosage not stated) were begun. The membranes ruptured spontaneously next day at 5 p.m. On the following day a rise of temperature occurred; non-hæmolytic streptococci were recovered from the vaginal swab, and penicillin and streptomycin (amounts not stated) were given. By the following morning the fœtal heart sounds had disappeared. A further vaginal swab was taken on this day, and also blood for culture; neither culture yielded any growth of microorganisms.

Labour began about 36 hours after the membranes had ruptured; pyrexia persisted throughout labour, the patient's temperature varying between 100° and 103·8° F., and her pulse rate between 88 and 140 per minute; her blood pressure varied between 135/75 and 160/100 mm. of mercury, but her urine remained normal.

Labour progressed normally; two rectal examinations were made. Five hours after labour began, low-forceps delivery of a stillborn macerated fœtus was performed under ether and oxygen anæsthesia. A second-degree perineal tear was repaired with catgut. The loss of blood *post partum* was "within normal limits".

Shortly after delivery the patient became severely shocked and was given intravenously "Methedrine", two pints of blood, and saline with noradrenaline and hydrocortisone (amounts not stated); but she died four hours later.

Post-mortem examinations were performed on the mother and the fœtus. The fœtus weighed 8 lb. 8 oz. and "showed evidence of anoxæmia and maceration due to generalized intra-uterine infection". The autopsy on the mother revealed clear yellow fluid in the peritoneal cavity, normal kidneys, some congestion in the liver and bilateral adrenal hæmorrhages. The cause of death was stated as adrenocortical failure due to septicæmia.

Comment.—This case is an example of bad ante-natal care in the first instance, and bad management in hospital in the second. At 35·5 weeks this patient had a blood pressure of 170/110 mm. of mercury, and she had gained 2 st. in weight during pregnancy. She was admitted to hospital, but discharged in two days because her blood pressure had fallen to "within normal limits". She should have been kept in hospital for the remainder of her pregnancy. The day after her readmission to hospital, vaginal examination with finger-tip dilatation of the cervix was performed. A total of seven such examinations, all with finger-tip dilatation, were made during the next 11 days. The patient subsequently developed septicæmia and died.

CASE 22.—This patient was a primipara, aged 35 years. She was first examined by her doctor at three months' pregnancy, when her general condition was good. Her home conditions were thought to be good also. So far as can be gathered from the records available, her pregnancy was uneventful up to 36 weeks. At this stage, as the vertex was not engaged, an abdominal X-ray examination was carried out.

She was admitted to a private hospital at term, in labour. Her general condition at this time was good. The head was engaged in the occipito-posterior position. In the course of the next 12 hours, labour progressed normally to full dilatation of the cervix. Three and a half hours later, low-forceps delivery with episiotomy was performed under "open ether" anæsthesia because the "mother's pains had gone off". A small post-partum hæmorrhage occurred, but was easily controlled by oxytocics. After delivery the patient complained of "wind and nausea". She was found to have a distended abdomen on the following day. The temperature and pulse rate remained within normal limits throughout labour, and continued to be normal until two days after delivery, when the temperature fell from 98·4° to 96° F. The lochia was excessive. On the second day after delivery, as the lochia was still excessive, 1 ml. of "Synkavit" was given. The episiotomy wound was noted to be inflamed, sloughing and offensive. On the same day penicillin therapy in a dosage of 500,000 units every six hours was begun, but the patient's condition deteriorated rapidly. Large purple patches were noted on the abdomen and limbs, towards midday she became cold and pulseless, and two hours later she died. No post-mortem examination was performed, and there is no record of either blood culture or culture of vaginal swabbings.

Comment.—This was a case of fulminating septicæmia. The Committee considered that it should be published as a warning that a patient may die very rapidly from such a condition, and that good aseptic technique in the management of obstetric cases is of paramount importance.

CASE 23.—This patient was a primigravida, aged 33 years, with a 12 years' history of sterility. She had good home conditions and regular ante-natal care, but there is no record that a pelvic assessment was made at any time during pregnancy.

At 42·5 weeks' gestation she was admitted to an obstetric teaching hospital in early labour. She was given castor oil, an enema and "Pitocin" (the dosage of the last-mentioned was not stated), as she was having "an inert type of labour". At this time her general condition was good—her pulse, temperature and respiration were normal and her urine was clear. The fœtal head was said to be "engaging", but no pelvic assessment was made. Twenty-six hours after the medical induction of labour, a vaginal examination showed that the cervix was dilated to admit four fingers. Surgical rupture of the membrane was then performed; the head was still unengaged at this time. The patient's general condition continued unchanged during the next 23 hours, but there was no advance.

At 11.20 a.m. on the third day—that is 60 hours after labour had started—lower segment Cæsarean section was performed, and a living male infant weighing 6 lb. 15·3 oz. was delivered. At operation a foul smell was noted; a peritoneal swab was taken and yielded a growth of *Staphylococcus pyogenes* sensitive to "Chloromycetin", "Aureomycin" and "Terramycin". A vaginal swab taken at the same time yielded *Staph. albus* sensitive to penicillin, "Chloromycetin" and "Terramycin".

After the Cæsarean section, the patient was given "Terramycin" intravenously for two days. Her temperature remained within normal limits until the evening of the fifth day after operation, when it rose to 102·4° F.; on that day and the day after she had some diarrhœa. On the morning of the sixth day after operation she appeared disorientated. There was a rise in her pulse rate, and her rectal temperature reached 107·2° F. A blood culture failed to yield any growth of organisms. Increasing peripheral circulatory failure occurred and did not respond to "Cortef", noradrenaline, fluids given intravenously or antibiotics. It is not known which antibiotics were given at this stage. She died after 22 hours of circulatory failure, and seven days after Cæsarean section following a 60 hours' labour.

Comment.—Whether this was a case of primary uterine inertia, or of disproportion, or of both, is not clear from the records supplied, as no pelvic assessment was made at any time. If there was disproportion, its cause was probably contracted pelvis, as the baby was not unduly large. The membranes were ruptured artificially 33 hours after the patient's admission to hospital in labour, though the head was still not engaged. There was, however, still no advance, and 33 hours after the rupture of the membranes, Cæsarean section was performed for fœtal distress. Intrauterine infection was evidently present at the time when the Cæsarean section was performed. The dangers of rupturing membranes in a primipara with inertia and an unengaged head are well known. The advantages are doubtful.

CASE 24.—This patient was aged 19 years. She had had one child two years previously. Nothing is known of her ante-natal condition in this, her second pregnancy. She had an emergency home confinement at about 28 weeks, and was delivered of an infant weighing 3 lb., who survived. Delivery was complicated by an intrapartum hæmorrhage, controlled by the administration of 1 ml. of "Pitocin" and 1 ml. of ergometrine. The placenta was retained for 80 minutes, and finally expelled in a "torn condition" by fundal pressure.

After delivery, the patient was transferred as an "emergency" to an obstetric teaching hospital. On her admission she was "pale and with a low blood pressure (98/52)". "Intermittent tachycardia, temperature and blood loss were noted during the next ten days." No details of treatment from the time of her admission until 12 days later were given. On the twelfth day a blood transfusion was given. During the administration of the second bottle of blood, her temperature rose to 100° F.; during the third she had a rigor, and her temperature reached 105° F. On the thirteenth day her temperature, taken four hourly, varied between 100° and 102·2° F. Her pulse rate remained at about 130 to 140 per minute. "Moderate blood loss" per vaginam occurred on the twelfth and thirteenth days. "The blood pressure never rose above 72/40." On the thirteenth and fourteenth days, 500 ml. of serum were given, and on the fourteenth day half a litre of 10% dextrose solution in water was given as well. On the same day the patient died. The records reveal that "she was jaundiced a few hours before death". Post-mortem examination revealed *Staph. aureus* septicæmia as the cause of death.

Comment.—Intrapartum hæmorrhage requiring "Pitocin" and ergometrine to control it was followed by a retained placenta, expressed 80 minutes later in a "torn condition" and almost certainly incomplete. On the patient's admission to hospital as an "emergency", one and a half hours later, she was "pale and with a low blood pressure (98/52)", obviously suffering from severe blood loss. Further blood loss occurred in the next 10 days, and a transfusion of three bottles of blood on the twelfth day indicates the severity of her anæmia. The intermittent tachycardia and rise in temperature suggest that she had infection of a retained placental remnant, and probably an infection due to the transfusion, as pyrexia and rigors occurred during the transfusion. Her pulse rate remained at about 130 to 140 per minute, a moderate vaginal loss continued, and her blood pressure remained low (72/40 mm. of mercury). Earlier exploration of the uterus and blood transfusion might have saved her.

This case illustrates the risk of infection during transfusion, and the need to deal with the causes of post-partum blood loss as well as its effects. The Committee considered that this was a preventible death due to *Staph. aureus* infection following blood transfusion for post-partum hæmorrhage, the result of an error in management.

CASE 25.—This patient was aged 30 years, and had a history of two miscarriages and one stillbirth. She had three living children, and had had toxæmia and a forceps delivery with the last pregnancy two years previously. When she was first examined by her doctor, she was noted to be "far from robust". Her home conditions were not known, but she did not look "prosperous". She was first seen, according to the available history, at 26 weeks of pregnancy, and at that stage her blood pressure was 160/76 mm. of mercury and she had "one-eighth" albumin in her urine. Her weight was not recorded. Her hæmoglobin value was 10·8 grammes per 100 ml., and her blood group was B, Rh-negative. No treatment was instituted at this visit, and she was not examined again for six weeks.

At the second visit, her blood pressure was 140/70 mm. of mercury, and there was a cloud of albumin in her urine; her weight was 9 st. 8 lb. Again no treatment was instituted. She next visited the doctor ten days later when her urine contained "one-quarter" albumin, her blood pressure was 160/70 mm. of mercury, her weight was 10 st. 2 lb., and she was œdematous. She was admitted to hospital the next day. The stated reason for her admission was that she lived a long way from hospital.

Ten days after her admission to hospital at 37 weeks of pregnancy, labour was induced by artificial rupture of the membranes followed by two doses of "Pitocin" (amount not stated) at hourly intervals. The reason given for the induction was that her condition, attributed to toxæmia, had deteriorated in spite of diet, rest and sedation. On the same day, her temperature was 103·4° F.; she was "very mucoid and semi-conscious and was given intranasal oxygen".

A low-forceps delivery was performed after a labour of 11 hours. She had a moderate post-partum hæmorrhage, and was given two pints of blood. At the time of delivery the presence of basal pneumonia was detected.

The day after delivery her condition was noted to have slightly improved. However, she had a cough, and was given penicillin (200,000 units every six hours) and streptomycin (0·5 gramme twice a day).

Her condition deteriorated during the night, and she died the next morning. The cause of death given was "septicæmia", with toxæmia of pregnancy as a contributing factor.

Comment.—This patient should have been admitted to hospital when she was first examined at 26 weeks, as she then had severe hypertension and albuminuria. Instead, she was not examined again for six weeks, and was not admitted even then, though she still had albuminuria. When she was seen 10 days after this, her condition had seriously deteriorated, and she was then admitted to hospital. Surgical induction of labour was apparently followed by basal pneumonia, which was the immediate cause of death.

Thrombophlebitis and Pulmonary Embolism.

There were 25 deaths from thrombophlebitis and pulmonary embolism. In seven of these cases thrombophlebitis of the leg had been diagnosed before embolism occurred.

CASE 26.—This patient was aged 30 years, and had three children. She visited her doctor for ante-natal care in this, her fourth pregnancy, at the seventh week. Her weight was 14 st. 6 lb., her blood pressure was 135/65 mm. of mercury, and her urine was clear. She was put on a reducing diet. Two weeks later her weight was 14 st. 3 lb., her blood pressure was unchanged and her urine was normal. One month later, at 13 weeks, her blood pressure was 120/70 mm. of mercury, and she had gained half a pound in weight. Her hæmoglobin value at this stage was 12·2 grammes per 100 ml. Her blood group was A, Rh-positive. In the next 12 weeks her weight gain was 3 lb., her blood pressure varied from 115/70 to 135/65 mm. of mercury and her urine was normal. When she was 24 weeks pregnant, she visited her doctor complaining of pain in her right calf, present for one week. On examination of the patient, varicose veins with thrombophlebitis were apparent. The doctor advised her to enter hospital for anticoagulant therapy, but she refused. He then treated her at home with procaine penicillin, 450,000 units per day, and visited her every day. Two days later, a red, tender swelling had developed in the region of the veins of the calf and extended half-way up the long saphenous vein. The patient said she would go into hospital if it was essential. On examining her, the doctor considered that the inflammation was more localized, and that she could stay in bed at home and continue with daily penicillin injections. Four days later there was marked improvement in the leg, but the following morning she suddenly became blue and breathless and died within three minutes. The stated cause of death was pulmonary embolism. There was no post-mortem examination.

Comment.—In the opinion of the Committee, this case history illustrates the importance of admitting to hospital all patients with venous thrombosis, especially when it occurs during pregnancy or the puerperium, so that anticoagulant therapy can be given under supervision.

CASE 27.—This patient was a primigravida, aged 28 years. She had good regular ante-natal care. Her total weight gain during pregnancy was 16 lb., her urine remained clear throughout, and her blood pressure never rose above 120/80 mm. of mercury. She was admitted to an obstetric teaching hospital at term, in labour. Normal delivery of a living child, after episiotomy, occurred eight hours after her admission. A "moderate" post-partum hæmorrhage complicated the third stage of labour, but was quickly controlled by the administration of "Pitocin" (1 ml.) and ergometrine (1 ml.). The puerperium was uneventful, and she was discharged from hospital on the tenth day.

Eleven days later she visited her doctor, complaining of pain and swelling of the left leg present for five days. On examining her the doctor found that there was a swelling of the saphenous vein extending up to the fossa ovalis, and that her temperature was 100° F. She refused to go into hospital or to rest in bed at home. Daily intramuscular injections of "Distaquaine Penicillin" were given, but anticoagulant therapy was withheld "on the grounds of inadequate supervision and the patient's refusal to enter hospital".

Six days later there was no pain or tenderness in the leg, but swelling was still evident. Three days after that—30 days after confinement—the patient suddenly collapsed and died of a pulmonary embolism.

Comment.—This case illustrates again the danger of venous thrombosis, and the need for hospital treatment under expert supervision. This life might have been saved but for the failure of the patient to cooperate.

Delivery with Complications (Exclusive of Hæmorrhage).

There were 24 deaths due to complicated delivery, including seven cases of rupture of the uterus. In 10 cases the complication was disproportion, which in two instances was associated with persistent occipito-posterior position of the fœtus, in two with shoulder presentation, and in one with breech presentation. In seven of these 10 cases labour was terminated by Cæsarean section. In two others, attempts at forceps delivery failed, and the patients died undelivered, one of them with ruptured uterus. There was one case of constriction ring in a possibly contracted pelvis, also terminated by Cæsarean section.

There were three cases of acute inversion of the uterus, and two cases in which rupture of the uterus occurred apparently without preceding obstruction.

The remaining nine deaths were due to various complications, as follows: air embolism, one; transfusion reaction, one; shock, one; precipitate labour and shock, one; forceps delivery and shock, one; intestinal obstruction followed by Cæsarean section, one; inhalation of vomitus during anæsthesia, one; twin pregnancy and rheumatic carditis, one.

In the majority of the cases of obstructed labour due to disproportion, there was no evidence that the possibility of disproportion had ever been considered by the medical attendant, and in only one instance was there evidence that the pelvic capacity had been assessed before labour began, or indeed before signs of obstruction appeared.

CASE 28.—This patient, aged 42 years, had had four normal confinements. There is no record of the weights of the babies. In this, her fifth pregnancy, she first attended her doctor for ante-natal care when about 12 weeks pregnant. She had phlebitis of leg varicose veins, and was in bed for one month. The only record of the ante-natal period available is that she was seen "monthly to the eighth month", and that her blood pressure was recorded monthly for five months, beginning when she was 12 weeks pregnant. At the first visit, the blood pressure was 142/90 mm. of mercury. According to the available information, she was never weighed. The urine was "normal" until about the thirty-fourth week of pregnancy, "when a cloud of albumin was found to be present". On this occasion her blood pressure was 160/90 mm. of mercury. (It had been 156/90 mm. of mercury one month before.) The patient was put to bed at home, with a salt-free diet. Bed rest continued for 17 days; at the end of this time her urine was free of albumin. Two days later labour began.

The patient was admitted to a private hospital, in labour, with a "show" at 10 p.m.; according to the nursing staff she was in normal labour. She was not examined by her doctor on her admission. Labour progressed with "strong pains" at intervals of 15 minutes. There is no record of any other observations on labour, or of examination at that time apart from the statement that the presentation was "vertex". "Strong pains" continued during the night. "Nembutal" (1·5 grains) was given at 2.30 a.m., and the membranes ruptured at 4.30 a.m. The dose of "Nembutal" was repeated.

At 6.30 a.m. the nurse in charge noted that the vulva was becoming swollen and discoloured. At 7 a.m. the doctor was informed, and visited the patient. On examining her, he found her "cyanosed, distressed and dyspnœic." The pulse was "almost impalpable". Vaginal examination revealed that the cervix was "fully dilated with the head high up". The doctor then decided to do a forceps delivery, under "open ether" anæsthesia administered by a colleague. The attempt at forceps delivery failed. "Coramine" was then given, and the patient was transferred by ambulance to a metropolitan obstetric teaching hospital. The time of her admission to hospital is not known, but she arrived in a moribund condition. Neither pulse nor blood pressure could be recorded; the heart rate was 88 per minute. "The uterus was enlarged to full time pregnancy, but the fœtal lie was not ascertained due to extreme obesity." Vaginal examination revealed the cervix "four fingers dilated, and the fœtus presenting as a posterior deflexed vertex above the brim". Fœtal heart sounds were absent. A "cut-down" in preparation for transfusion was done, and "Coramine", "Methedrine", oxygen and aminophylline were given; but the patient died undelivered one hour after her admission to the teaching hospital. Post-mortem examination revealed a ruptured uterus.

Comment.—This case illustrates the possibility of obstructed labour from disproportion even in a multipara who has had several normal deliveries, and the importance in ante-natal history-taking of recording the weight of the previous babies, especially the heaviest one, the method of delivery, and the condition at birth.

In this case the possibility of disproportion does not seem to have been suspected, particularly as the doctor did not think it necessary to see the patient on her admission to hospital or during the night when she was in strong labour. It seems, too, that the nurse did not call the doctor's attention to the failure of the head to descend into the pelvis, in spite of "strong pains" every quarter of an hour for nine hours. The high application of forceps in an attempt to deliver the baby is dangerous, and not in accordance with modern practice.

This history illustrates the dangers which may occur when a patient is admitted to hospital during the night, and is not seen by her medical practitioner, who may rely on inadequate information provided by less qualified personnel. This was a preventible death due to lack of appreciation of the situation by both nurse and doctor.

CASE 29.—This patient was aged 24 years, and had had two previous confinements (no record of the previous birth weights or of any abnormalities was available). She did not attend her doctor for ante-natal care until she was approximately 30 weeks pregnant. On this occasion her blood pressure was 125/70 mm. of mercury, her urine was normal and her weight was 8 st. 9 lb. She attended again only once, two months later, when her blood pressure was 130/70 mm. of mercury, her urine was normal, and her weight was 9 st. 3 lb. On this occasion the position was thought to be right occipito-posterior, and the fœtal heart was heard.

The patient was admitted to hospital in labour five days later. The membranes had ruptured at home—the time is not known. Her condition was good. She had had strong contractions at home, but these went off on her admission to hospital. The presentation, which later proved to be by the shoulder, was thought to be by the vertex, right occipito-anterior. Her temperature was normal and her pulse rate 84 per minute, but her blood pressure was not recorded. The fœtal heart sounds were audible.

By 8.30 a.m. next day, the uterine contractions had returned (there is no record of how long they had been present), and she lost 4 oz. of bright red blood per vaginam. By 9.15 a.m. the cervix was fully dilated; the uterine contractions again went off, and this time the fœtal heart could not be heard. Her general condition began to deteriorate. She rapidly became shocked, and "very little anæsthetic (ether)" was used while high-forceps delivery was attempted after correction of the shoulder presentation to a vertex. This failed, and the baby was found to have escaped into the mother's abdominal cavity through a rupture of the uterus. It was finally delivered by the breech. During this time the anæsthetist had been attempting to resuscitate the patient with adrenaline (1 ml.) and half a bottle of serum given intravenously. She died at 10 a.m. No post-mortem examination was performed.

Comment.—This patient died from shock due to rupture of the uterus following shoulder presentation, which was not recognized early in labour.

CASE 30.—This patient was a primipara, aged 28 years. Her ante-natal care was regular from the third month of pregnancy. Visits were made monthly to the twenty-eighth week, fortnightly to the thirty-second week and weekly thereafter. Her blood pressure remained steady at 130/80 mm. of mercury until she was about 38 weeks pregnant. At this stage it rose to 150/100 mm. of mercury; by the next week it had fallen to 140/90 mm. of mercury and it remained at this level during the next two weeks. She gained 4 lb. in weight in one week at this time, and had œdema of the feet, but no albuminuria. No active treatment is mentioned; three days before labour started, her blood pressure was 150/90 mm. of mercury.

She was admitted to a metropolitan private hospital in labour two weeks after the expected date of confinement. On her admission she was having weak contractions, which continued at irregular intervals for the next two days. On examination of the patient, the doctor found the fœtus lying "a bit posterior". On the third day after her arrival in hospital, the patient began to have stronger contractions. When she was seen by her doctor, he considered that "it was a fairly obvious posterior position"; but as she was "slowly progressing" and the pelvis was "reasonable", "he expected the baby to turn".

There is no mention of a pelvic examination or assessment having been made, either in pregnancy or at any time in labour before the second stage.

Later that evening, the uterine contractions were much stronger, and the doctor noted that "there was no vulval pressure". This was the reason he gave for deciding to apply forceps and to deliver the baby as a "persistent occipito-posterior".

After preliminary digital stretching of the perineum, the forceps was applied under "open ether" anæsthesia, and strong traction was used without success. The forceps was then removed and the doctor made a vaginal examination with a view to "disimpacting the head and rotating it to an anterior position".

On vaginal examination, he discovered a prolapsed cord with no pulsation. Thinking that if he delivered the child quickly it might have a chance of survival, he reapplied the forceps with the occiput still in the posterior position; strong traction was again used and the forceps came off and caused a third-degree perineal tear; the forceps was then reapplied and the infant delivered. The doctor then turned his attention to the infant, and spent 20 minutes attempting to resuscitate it. During this time the mother lost a lot of blood, although "Pitocin" and ergometrine had been given. The doctor states that when he realized that he could do nothing further for the infant, he noticed how much blood the mother was losing; the actual amount is not stated. He then proceeded to repair her perineum under "open ether" anæsthesia given by the night sister.

On completion of the repair operation, the patient was noticed to be very pale, but she had "quite a good pulse". Blood transfusion was considered, but not given, because the patient's condition appeared to be improving. The night sister stayed with the patient during the next four hours, and stated that although she was "drowsy and weak" she took some fluids, and her condition was "quite moderate". The night sister then left the patient for a few minutes, and on returning found her dead. No post-mortem examination was performed.

Comment.—The Committee considered that in a case such as this, as the patient was a primigravida aged 28 years and the fœtus was in the occipito-posterior position, a consultant should have been called before the decision was made to deliver the baby with forceps as a "persistent occipito-posterior", especially in the absence of vulval pressure. A blood transfusion should have been given.

CASE 31.—This patient was aged 30 years, and in her fifth pregnancy. She had had three full-time normal confinements; the last child had died from atelectasis three days after it was born. She had also had one miscarriage three years before this pregnancy. She lived in an isolated country district, where there was a small hospital with only one trained nurse on the staff. She had regular antenatal care from her doctor, and had a normal and uneventful pregnancy. Her urine was normal throughout, her total weight gain was 1 st. 1 lb., and her blood pressure averaged 125/80 mm. of mercury. The hæmoglobin value at seven months was 11 grammes per 100 ml. Blood grouping was not done. There is no information concerning previous birth weights.

The patient was admitted to hospital at or about term in labour. On her admission, the vertex was found to be presenting in the left occipito-posterior position (not engaged); "a small amount of head was fitting into the pelvis". The fœtal heart sounds were present and the uterine contractions were strong.

Labour began at 10 p.m. on that day, and the membranes ruptured at 5 a.m. the next day, seven hours after labour had started. The patient was anxious and restless all the time she was in labour because she had lost the last child. At 5 a.m. a vaginal examination under ether anæsthesia was made by the doctor in charge of the case, and the cervix was found to be fully dilated. The forceps was applied once, "mid to high", without success. The head was then rotated, and another unsuccessful attempt at delivery was made.

At 5.30 a.m. the uterine contractions were strong, and the vertex was still in the left occipito-posterior position. At 5.45 a.m. the patient recovered consciousness and was given 50 mg. of pethidine.

Labour continued until 7.30 a.m. The fœtal heart could not be heard at this time, and the patient's condition began to deteriorate. The pulse rate began to rise. She collapsed at 8 a.m. The intravenous administration of "Dextran" was commenced, but she died ten minutes later, undelivered. Post-mortem examination revealed a ruptured uterus. The tear was on the anterior surface near the fundus, "slightly on the right side", and 3·5 in. long.

The doctor in charge of this case was in an isolated country town, and no expert assistance was available, although he tried to get it.

Comment.—In the absence of any information regarding the weight of previous babies or the weight of the present one, it is not possible to determine whether contracted pelvis was the primary cause of the difficulty, though this is suggested by the statement that the fourth child died of atelectasis three days after its birth. The medical attendant, realizing that he was dealing with a difficult case, tried to get expert help, but as he worked in a remote country area, none was available. This has now been remedied, and consultants can be called to all parts of New South Wales.

Abortion.

The total number of deaths from abortion was 73; of these 56 were the result of criminal interference, 15 apparently followed spontaneous abortion, and two followed abortions induced for medical reasons.

Infection was the cause of death in 53 cases; in two of these the uterus had been perforated. The most common infecting organism was *Clostridium welchii*, but in one case it was *Cl. tetani* and in four *Staph. aureus*.

Twenty patients died from other causes than sepsis, as follows: hæmorrhage, seven; air embolism, four; shock due to injection of fluid into the uterus, four; pulmonary embolism, one; congestive cardiac failure, one; anæsthesia during curettage, one; anuria without sepsis, one; unknown, one.

Altogether there were 18 deaths from renal failure and anuria, 17 of these occurring in the "infection" group. All the deaths from anuria occurred in obstetric hospitals.

The social status of the 73 patients who died was as follows: married, 50; single, 17; separated, one; not known, five.

On reading the records of these fatal cases, one is impressed with the frequency with which the patient was discharged from hospital within a day or two after dilatation and curettage of the uterus, even when there had been pyrexia at the time when the operation was performed. In some instances she was readmitted to hospital shortly after discharge, in a serious condition from septic peritonitis and other manifestations of active septic infection.

In several cases dilatation and curettage were done almost immediately after the patient's admission to hospital and without allowing sufficient time for observation and proper assessment of the patient's condition, particularly with regard to the possible presence of pelvic peritonitis. Too often dilatation and curettage were done without any urgent indication such as dangerous bleeding, when the presence of abdominal pain and tenderness with or without pyrexia might have aroused suspicion of this complication, and counselled delay until after temperature and pulse rate had returned to normal and all signs of spreading sepsis had subsided.

It is, perhaps, not generally appreciated that when in an incomplete abortion curettage has to be done, the safest and most effective curette is often the operator's forefinger, aided by downward pressure on the fundus by the external hand, rather than a sharp or blunt instrument, which must necessarily be used more or less blindly inside the uterus, with the result that, as happened in some of the recorded cases, quite large pieces of placenta are left attached to the uterus and cause severe continuing hæmorrhage.

CASE 32.—This patient, aged 30 years, had had two children and three abortions. She was admitted to a metropolitan hospital with incomplete abortion. She complained of lower abdominal pain, and slight vaginal bleeding was present. Her temperature was 98·6° F. and her pulse rate 76 per minute; vaginal swabs yielded a profuse growth of *Staph. aureus*.

Dilatation and curettage were done three hours after her admission, and placental débris was evacuated. On

the following day the patient complained of vague abdominal pain and vomited, and tenderness was present in both loins. Her temperature was 100° F. and her pulse rate 132 per minute. With analgesics the pain and tenderness subsided by the fourth day after operation. Her condition remained "satisfactory" till two days later (the sixth day after operation), when she woke with a sudden pain in the left side of the chest, collapsed and died. Post-mortem examination revealed a pyæmic lung abscess.

Comment.—Though the temperature and pulse rate were not abnormal on her admission to hospital, the lower abdominal pain should have suggested the possibility of pelvic peritonitis or septic thrombophlebitis, which, in the absence of any urgent indication such as severe uterine hæmorrhage, should have contraindicated active interference, especially as soon as three hours after admission to hospital. The death was certified as due to chronic myocarditis, but the Committee could not find any evidence of this. In its opinion death was due to septic abortion and pyæmia.

CASE 33.—This patient, aged 36 years, was admitted to a metropolitan hospital with incomplete abortion, and a history of having passed a fœtus one week before. There is no record of previous pregnancies or abortions, if any.

On her admission to hospital her temperature was 100° F., and her pulse rate 76 per minute. She had a brown vaginal discharge, "moderate" pain in the lower part of the abdomen and tenderness in both vaginal lateral fornices; the cervix was dilated to admit one finger.

The uterus was curetted on the day of her admission, and placental tissue was removed. No vaginal cultures were taken, but penicillin and streptomycin were administered (doses not stated). A blood count showed that the erythrocytes numbered 2,910,000 and the leucocytes 38,000 per cubic millimetre, but the hæmoglobin value was not recorded. No blood transfusion was given. The patient died two days after operation; but there is no record of her condition between the operation and her death, and there was no post-mortem examination.

Comment.—Because of scanty records and no post-mortem examination, it is not possible to say what the cause of death was. It is, however, clear that there was no indication for urgent curettage on the day of the patient's admission to hospital, without any time for observation, as there was no serious bleeding, and that the operative intervention was contraindicated by pyrexia, lower abdominal pain and tenderness in both lateral fornices, all suggesting the presence of septic peritonitis.

CASE 34.—This patient, aged 22 years, was admitted to a metropolitan obstetric hospital with threatened abortion at 24 weeks, caused by falling against some steps at her home on the day before. Her condition on admission was said to be good; moderate hæmorrhage and slight pain in the abdomen were present, but there is no record of pulse rate, temperature or hæmoglobin level. On the day of her admission she passed the fœtus, and two hours later the uterus was curetted. She was discharged on the fourth day after operation, as she was "clinically satisfactory", though again there are no records of pulse or temperature. She was told to stay in bed for a week and have a check by her own doctor.

Eighteen days later she was readmitted to hospital with vomiting and a history of rigors since the fourth day after discharge and of diarrhœa for a week. Examination showed signs of pelvic peritonitis. She died 10 days after her admission, having failed to respond to antibiotics. Post-mortem examination revealed pelvic peritonitis with pus formation and adhesions, and purulent pericarditis.

Comment.—This case illustrates the danger of curettage of the uterus for no apparent urgent reason such as dangerous bleeding, and especially in the presence of abdominal pain and without any evidence that pulse rate and temperature were normal. It also exemplifies the risk of discharging a patient from hospital soon after curetting her uterus (in this case on the fourth day), and especially without excluding the possibility of commencing spreading sepsis. The presence of purulent pericarditis indicates blood-stream infection, which may or may not have been caused by the curettage.

Ectopic Gestation.

Fifteen deaths from ectopic gestation were investigated; eight were considered preventible and seven non-preventible. Three resulted from non-cooperation by the patient, such as denying the possibility of pregnancy.

The causes of the deaths were: hæmorrhage, six; sepsis, three; pulmonary embolism after operation, three; cardiac arrest under anæsthesia, two; post-operative intestinal obstruction, one.

Sepsis occurred in eight cases, severe internal hæmorrhage occurred in 12. Of 15 patients, eight received blood transfusions, two received serum or saline infusions, and the remaining five had neither.

There was a delay varying from a few hours up to 21 days before some were operated on after being examined by a doctor, the diagnosis being missed for periods of up to 21 days. Three patients were not operated on at all, and inexplicable delay occurred in some cases while transfusions were being given in an attempt to restore the patients' condition before operation, the internal hæmorrhage going on in the meantime.

One woman died in the ambulance on the way to a distant obstetric hospital, passing several general hospitals on the way. There was one case of seven and a half months' abdominal pregnancy associated with severe toxæmia and hypertension; this patient died from hæmorrhage after operation.

The question of blood transfusion in ruptured ectopic pregnancy has often been discussed by the Committee. It would seem that massive transfusion before the bleeding points are secured may be attended by considerable danger of starting fresh hæmorrhage; that if the patient is fit to stand a general anæsthetic without it, transfusion should not be given till bleeding has been controlled; and that if, because of extreme exsanguination, she is considered unfit to stand the anæsthetic, only enough blood should be given to make her fit, the complete transfusion being started as soon as bleeding points are secured.

Cerebral Hæmorrhage.

There were nine deaths from cerebral hæmorrhage. In seven of these the hæmorrhage was said to be due to rupture of an aneurysm. All the deaths occurred after delivery, the intervals elapsing between delivery and the fatal hæmorrhage being as follows: 3, 5, 12 and 24 hours; 10 and 12 days; and 6 weeks. One of the patients had suffered from frequent attacks of migraine for years.

In two cases the cerebral hæmorrhage was due to chronic hypertension. In one of these the blood pressure was 250/160 mm. of mercury in early pregnancy, and albuminuria was present. Fatal cerebral hæmorrhage occurred at the fourth month of pregnancy, three days after the patients' admission to hospital for therapeutic termination. In the other the blood pressure was 200/125 mm. of mercury at the second month, and the cerebral hæmorrhage occurred at the thirtieth week. The patient had refused termination of pregnancy and had good ante-natal care, mostly in hospital.

It is obvious that in cases of chronic or acute hypertension of this magnitude, cerebral hæmorrhage is always a possibility. Admission to hospital and the use of hypotensive drugs may in future enable pregnancy to be safely continued.

Other Causes of Maternal Deaths.

Twelve maternal deaths were due to causes not included in any of the foregoing categories. These were as follows: (i) Acute pyelonephritis. (ii) Shock from injection of fluid into the uterus, while the patient, on her doctor's advice, was douching herself for vaginal irritation. (iii) Amniotic embolism three hours after normal delivery. There was no unanimity among the pathologists who examined the lung sections as to the presence of amniotic emboli. (iv) Air embolism on the seventh day after delivery, while the patient was in the knee-chest position doing post-natal exercises. (v) Mid-forceps delivery and third-degree tear; death occurred 80 minutes post partum from obstetric shock. (vi) Ruptured uterus at 32 weeks, probably from a fall. (vii) Sudden death on the tenth post-partum day during blood transfusion for severe (Osler's) anæmia.

(viii) Hydatidiform mole. (ix) Hysterotomy and sterilization for psychosis, at the fourth month of pregnancy; sudden death occurred seven days later. (x) Cardiac infarction four hours post partum. (xi) Retroperitoneal hæmorrhage (afibrinogenæmia). (xii) Puerperal mania.

Attention is drawn in this section to the danger of vaginal douching during pregnancy, as illustrated by the second case listed above.

Maternal Deaths Associated with Anæsthesia.

Nine deaths associated with anæsthesia were investigated by the Committee during the seven-year period covered by this report. Of these, seven were considered to be due to the anæsthetic, including one in which the cause of death was inhalation of vomitus. The anæsthetic given in this case was ethyl chloride and "open ether" for forceps delivery; bronchoscopy, suction and cardiac massage were of no avail.

Cæsarean Section.

Thirty deaths following Cæsarean section were investigated by the Committee during the seven years covered by this report. Twenty-seven of these deaths were classified as maternal and of these 22 were considered to be preventible.

In some cases the Committee considered that, on the evidence supplied, there was insufficient reason for performing the operation. In other cases technical difficulties were encountered, and the patient might have been saved if consultant help had been sought beforehand. Other cases illustrate the importance of having blood available for transfusion before the operation is started, so that replacement therapy can be begun with the minimum of delay should the need arise.

Three deaths followed Cæsarean section for chronic pyelonephritis, intestinal obstruction and cerebral thrombosis respectively. These were classified as "non-maternal" deaths.

CASE 35.—This patient was a primigravida, aged 24 years. She visited her doctor for ante-natal care regularly. He, however, kept no records, and the only available information covering the ante-natal period is that she was examined regularly "monthly to the seventh month, and fortnightly to the ninth month". Her urine was examined "regularly", but there is no information as to the results of these tests. She was weighed, but there are no records of the results. The blood pressure "was never over 140/90". A pelvic examination was made at some stage of the pregnancy (date unknown), but no measurements were recorded. The doctor stated that "she had a justo-minor pelvis".

At 40 weeks she had a "show", and was admitted to hospital in labour at 9.30 p.m. next day. On her admission, the vertex was presenting in the left occipito-anterior position, and the head was not engaged. The fœtal heart rate was 120 per minute, and the patient's condition was "good". She was having "pains every ten to fifteen minutes—membranes intact".

No further details of her condition during labour are available. Fifteen hours after her admission, with the membranes still intact, lower segment Cæsarean section was performed for "arrest of descent of the fœtal head about mid-pelvis, and contracted outlet". No vaginal examinations were made at any time during labour. There was no information as to whether rectal examinations were made. It is also not known whether there was fœtal or maternal distress, or how much dilatation of the cervix had occurred before the decision to perform Cæsarean section was made.

A moderate post-partum hæmorrhage was controlled by "Pitocin" and ergometrine (dosage not stated); "Pitocin", 1 ml., was given every six hours.

The patient's progress was satisfactory during the 24 hours after operation. On the next two days, vomiting of bile-stained fluid was "troublesome". On the fourth day after operation, jaundice developed and also "severe right-sided thoracic pain".

Treatment included the administration of penicillin (500,000 units per day), started the day after operation, "Prostigmin" (1 ml. on the third day and 0·5 ml. per day thereafter), "Andramine" (two tablets three times a day), and also sedative mixtures of bismuth as well as potassium bromide and chloral. No fluids were given intravenously until the day before death, although the patient was vomiting frequently. By the evening of the fifth day she was "feeling hot", and the sister in charge found her skin "cold and clammy". Eight bowel actions with the passage of fæcal blood-stained fluid had occurred during that day, and the abdomen was distended. The following day, the sixth after operation, diarrhœa and vomiting of "brown fluid and mucus" were persistent, the pulse was weak and rapid, and the skin was cold and clammy. One bottle of 5% dextrose solution in normal saline was given intravenously, and that was all. On the seventh day after operation her condition steadily deteriorated. "Wangensteen's tube was running from 1 p.m." She died at 4.50 p.m. that day. No post-mortem examination was performed. The cause of death was stated on the death certificate to be (*a*) Cæsarean section, (*b*) acute myocarditis.

Comment.—It was noted that the vertex was not engaged at the beginning of labour though the patient was a primipara, that the pelvis was diagnosed as generally contracted, and that presumably a test of labour was given. There was, however, no evidence that the test was adequate, or that it had failed, especially in view of the fact that the Cæsarean section was done while the membranes were still intact. The Committee considered that the cause of death was septic peritonitis following a Cæsarean section that was probably unnecessary.

CASE 36.—This patient was a primigravida, aged 21 years. Details concerning her ante-natal care are very scanty, as her doctor did not keep any records and failed to provide any information when it was requested. It is, however, known that the patient visited him for ante-natal care on three occasions, and probably attended at other times as well. There is no information concerning the results of these examinations.

The patient was admitted to the local public hospital for treatment of preeclamptic toxæmia shortly before term, but was discharged again the next day. Five days later she was readmitted to hospital for Cæsarean section. The indication for operation as stated was "pre-eclamptic toxæmia". Delivery of a living child by Cæsarean section was effected the following day.

She remained in hospital for 12 days after delivery. During this time her temperature ranged from 99° to 100° F. "Distaquaine penicillin" (300,000 units twice a day) and sulphadiazine (two tablets every four hours) were given for four days. The pyrexia did not respond to this treatment; it is not known if any other treatment or investigations were carried out. Her temperature was 100° F. on the evening before she left hospital.

Subsequently she was readmitted to hospital "several times" with "tachycardia" for which no cause was found. On the twentieth day of the puerperium she suddenly collapsed while on the X-ray table.

The cause of death was stated to be "cerebral embolism". No post-mortem examination was performed. Owing to lack of cooperation on the part of the doctor, and inadequate hospital records, no further information is available.

Comment.—This case is an example of bad ante-natal and post-operative care. The patient was admitted to hospital with a diagnosis of preeclamptic toxæmia, yet was discharged on the following day for no apparent reason. She was readmitted five days later, and a Cæsarean section was done next day, although there is no evidence that the operation was necessary or indicated. She developed sepsis after operation, and was discharged on the twelth day of the puerperium in spite of the fact that the temperature had reached 100° F. on the previous evening. No post-mortem examination was done, and there is no real evidence that death was due to cerebral embolism. The Committee considered that death was due to sepsis following a probably unnecessary Cæsarean section.

CASE 37.—This patient was aged 39 years. Her first pregnancy had ended in lower segment Cæsarean section for "failure to progress after trial labour". During the second pregnancy, three years later, she was "grossly overweight", and had preeclamptic toxæmia, for which she was confined to bed for six weeks before delivery. Delivery was again by lower segment Cæsarean section after an unsuccessful trial of labour for disproportion.

She thus embarked on her third pregnancy with a history of two previous Cæsarean sections, and toxæmia. She was "overweight and inclined to flabbiness", She was

not weighed during pergnancy. She was very well up to the eighth month, and on a reducing diet. The doctor who had supervised her previous pregnancies then left the district, but before leaving he advised his successor to perform an elective Cæsarean section in view of the patient's past obstetric history. At 36 weeks her blood pressure was 140/75 mm. of mercury and her urine was normal. She was not weighed, but was given a diet of low salt, low carbohydrate and high protein content. At two subsequent ante-natal visits, the blood pressure reading was 145/70 mm. of mercury and her urine was still normal. Two weeks before the expected date of confinement, she was admitted to hospital for Cæsarean section.

On her admission, her general condition was "good", but she had mild toxæmia of pregnancy with slight œdema of the ankles and fingers. The vertex was presenting in the left occipito-anterior position and was not engaged, and the fœtal heart sounds were present.

On the day after her admission, a lower segment Cæsarean section was performed with difficulty. Numerous adhesions and large sinuses over the anterior wall of the uterus made the operation difficult. Considerable intrapartum hæmorrhage occurred. On her return to the ward, she was given a blood transfusion (amount not stated) followed by 1 litre of dextrose-saline solution. After this treatment her condition was satisfactory until the next day. On this day she complained of abdominal pain and discomfort, and her abdomen was hard and tender. Penicillin (1,000,000 units twice a day) was given and continued for five days. On the second day after operation, paralytic ileus developed with "possible partial bowel obstruction". Treatment was as follows: the continuous intravenous administration of fluids (dextrose-saline solution and Darrow's solution) with 250 mg. of "Terramycin" in the flasks; insertion of a Wangensteen's tube for suction; the administration of "Parenamine" and 600,000 units of gas-gangrene anti-serum, and supplementary vitamins by intramuscular injection. No bowel action occurred after operation, and enemata were given on three occasions, two days, three days and six days after operation, the only result being the return of coloured fluid containing flecks of fæces. A gradual rise of temperature occurred, from 99·5° F. on the third day after operation to 101·6° F. on the sixth. On the fifth day basal pneumonia developed, and on the sixth the patient was transferred to the district hospital.

Treatment after her transfer to the district hospital was similar to that at the maternity hospital. Her temperature fell from 101·2° F. on the sixth day to 98·4° F. on the eleventh day. Her pulse rate also fell from 100 to 88 per minute. On the tenth day after operation the myocardium began to fail, and digoxin (0·5 mg.) was added to the fluids given intravenously. At 2 a.m. on the eleventh day, her pulse rate rose to 128 per minute; she became unconscious at midday and died at 4 p.m. No post-mortem examination was performed.

Comment.—The Committee considered that a medical practitioner of only four years' standing taking over the care of this patient at 36 weeks' pregnancy was rightly advised that a third Cæsarean section should be performed. Numerous adhesions and very large vessels were found, and resulted in great technical difficulties and severe hæmorrhage during the performance of the lower segment operation, which contributed to the subsequent death from intestinal obstruction. Among the contraindications for a lower segment operation are dense adhesions and large vessels. An upper segment operation under such circumstances is often the wiser course. This patient might have survived, had she been transferred to a large district hospital and been operated on by an experienced consultant called in for the purpose. The case also illustrates some of the disadvantages of elective lower segment Cæsarean section, in contrast to the same operation performed after labour has been in progress for even a short time. If the patient has been in labour for some time, less bleeding is likely to be encountered, as the lower uterine segment is stretched and more or less thinned out and relatively non-vascular. For the same reason, the operation area is more accessible and the operation easier. There is, too, less chance of unexpectedly delivering a premature baby through the patient's being mistaken in her menstrual dates.

CASE 38.—This patient was aged 35 years, and had had one child by forceps delivery; the birth weight was 7 lb. She had also had a miscarriage. The ante-natal care in this (third) pregnancy was given at the out-patient department of a public obstetric hospital; her blood pressure, urine and weight were normal throughout. Pelvic examination revealed "adequate findings", but it was recorded that the head did not engage.

The patient was admitted to hospital at 5.30 p.m. in labour at term. The vertex was presenting, reported to be in the left occipito-anterior position, but not engaged. The fœtal heart was heard. Fourteen hours later, at 7.30 the next morning, the cervix was thought to be fully dilated, and an attempt at high-forceps delivery was made by the resident medical officer. The indication given for the forceps delivery was "no progress". The attempted forceps delivery failed, and it was discovered that in fact the cervix was not fully dilated; the anterior lip could still be felt.

Labour was allowed to continue after this. Although the contractions became violent, there was no progress.

At 10 a.m., pulse and blood pressure readings indicated that the patient was becoming shocked. Intravenous therapy was started at this time, and she was given five pints of blood. "Pot. brom. and chloral and pethidine (one or two doses) were given" during labour.

Labour was allowed to continue, in spite of a diagnosis of obstructed labour, until 4.10 p.m. that day, when lower segment Cæsarean section was performed by a consultant obstetrician. A stillborn baby weighing 8 lb., in the persistent occipito-posterior position, was delivered with much difficulty.

Two and a half hours after operation, the patient suddenly became cyanosed and shocked, and a diagnosis of pulmonary embolism was made. The diagnosis was confirmed radiologically. Heparin was given intravenously, and "Methedrine", digoxin and antibiotics were given, but the patient died within 17 hours. There was no post-mortem examination.

Comment.—The Committee considered that an attempt at high-forceps delivery, even though the os is fully dilated, is unjustified in any circumstances. It is obvious that the posterior position of the occiput was not recognized. The presence of violent uterine contractions without progress, especially when followed by signs of shock, should have led to an immediate reassessment of the position by a consultant, instead of which the patient was left for six hours before Cæsaren section was done for obstructed labour.

CASE 39.—This patient, aged 47 years, had had eight previous confinements, all of which were said to be difficult, but there is no information regarding the weight or condition of the babies. The last had been born eight years before; in addition, there had been mild toxæmia with the last three pregnancies. During the present pregnancy she had cystitis early, but no other abnormality was noted until labour started at term, when the head was not engaged and the shoulder was said to be four fingers' breadth above the symphysis. Labour began at 10 p.m., and the membranes ruptured at 4 a.m. next day. Fifteen minutes later, while the head was still high, forceps was applied "as trial of forceps". As no advance was made, the attempt was abandoned without any heavy traction, and it was decided to deliver her by Cæsarean section. A lower segment Cæsarean section was performed immediately under "Pentothal", nitrous-oxide, oxygen and ether anæsthesia. Hæmorrhage at and after delivery was excessive, but a transfusion of 1·5 pints of blood restored the patient to a satisfactory condition. The operation was completed at 8 a.m. The baby's birth weight was 8 lb. 6 oz.

At 9.30 a.m. her condition deteriorated, and a further transfusion was given. After the next half pint of blood had been given she improved, but again deteriorated, and attempts to speed up the rate of flow of blood were followed by deterioration of her condition, with an increase in moist sounds in the chest. Death occurred at 12.30 p.m., four and a half hours after the completion of the operation.

A complete post-mortem was not carried out, the abdomen only being opened. There was a considerable amount of black blood present, mixed with peritoneal exudate.

Comment.—The high application of forceps is not justifiable in any circumstances, and especially a quarter of an hour after spontaneous rupture of the membranes. Death was due to hæmorrhage during and after Cæsarean section, the necessity for which is doubtful, as it was done two hours after spontaneous rupture of the membranes and without evidence of an adequate indication, the total duration of labour being eight hours.

CASE 40.—This patient was aged 34 years, and had had one child seven years before; the labour was said to have been long and difficult. The child had suffered from

"cerebral palsy", and died in infancy. When she was first examined in this pregnancy, she was four months pregnant. There are few records of her ante-natal care; the only available information is that her pelvic measurements were "within normal limits". She was weighed each month and the gain was said to be normal, but her weight was not recorded. Her urine was tested every month, and "no abnormality was detected". Her blood pressure was "estimated but not recorded at each visit, but was within normal limits". She was examined monthly to the eighth month and fortnightly to the ninth month. There is no record of blood grouping, hæmoglobin estimation or Wassermann or Kahn tests.

At term she was admitted to hospital with a slight "show" and slight pains, which continued throughout the day. She was kept in hospital, although not really in labour, because of serious floods in the district.

On the twelfth, thirteenth and fourteenth days after her admission she had only slight contractions. On the evening of the fourteenth day she was given 5 grains of quinine sulphate, and subsequently had contractions at five-minute intervals during that night. The membranes ruptured at 3·15 a.m.; her doctor examined her at 6 a.m. and found her distrissed with contractions strong and frequent since 3.15 a.m. The position was left occipito-posterior and the vertex was not engaged, and he noted a Bandl's retraction ring rising and decided on Cæsarean section.

There are no records of the temperature, pulse or blood pressure during labour, or of any vaginal or rectal examinations.

At 8 a.m. lower segment Cæsarean section was performed, and a living child weighing 8 lb. 10 oz. was extracted. Immediately after delivery of the infant the patient became shocked, and collapsed with dilated pupils and a weak pulse. The anæsthetic used with nitrous oxide, oxygen and ether.

Considerable hæmorrhage occurred at operation, and the patient was returned to the ward in a collapsed condition. One and a half hours later she was given intravenously serum (500 ml.) followed by 1 litre of 4% dextrose solution in N/5 saline. Her pulse rate and temperature were recorded hourly; the temperature varied between 97° and 99·2° F., and the pulse rate ranged from 74 to 100 per minute; at times the pulse was imperceptible.

On intravenous therapy her general condition improved, and the drip administration of fluid was stopped at 12 midday. Penicillin (1,200,000 units) was given in three doses after operation.

By 2 p.m. her condition had again deteriorated, and she was given serum albumin, 200 ml. twice—each 200 ml. being followed by 1000 ml. of 5% dextrose solution in water. Finally, in the late afternoon or evening (the time is not stated), she was given 300 ml. of blood and a further 1000 ml. of 4% dextrose solution in N/5 saline. Another 550 ml. of blood were given intravenously and morphine, one-sixth of a grain, was given intramuscularly at or about the time when she had a further hæmorrhage per vaginam.

During that night she was "rational but pulseless at times". The next morning her condition was still poor, and the extremities were noted to be cold. A further 500 ml. of 5% dextrose solution were given, but her condition deteriorated steadily, and she died at 2 p.m. that day. The causes of death set out on the death certificate were as follows: cardiac failure, surgical shock, difficult labour due to pelvic disproportion, hæmorrhage at Cæsarean section. No post-mortem examination was performed.

Comment.—In view of the bad obstetric history, and of the death of the first and only child (born seven years before) with cerebral palsy, which could have been due to birth injury, it would have been wiser to refer this patient to a consultant early in pregnancy for special care in delivery. Delivery this time was complicated by posterior position of the occiput, and probably by disproportion. Cæsarean section was obviously necessary, and might with advantage have been done sooner. The cause of the "considerable hæmorrhage" and collapse at operation is not clear. The blood transfusion was too late and too little (850 ml.). It is always advisable to have advance preparation made for transfusion before starting Cæsarean section.

"Non-Maternal" Deaths (By International Classification).

There were 57 deaths that were classified as non-maternal.

The causes of death in these cases were as follows: valvular heart disease, 11; congenital heart disease, one; chronic myocarditis, one; coronary occlusion, three; cerebral embolus following mitral stenosis, one; paralytic ileus after termination of pregnancy for chronic hypertension and appendicectomy, one; cerebral hæmorrhage associated with chronic hypertension, three; pulmonary embolism of unknown origin (no autopsy), one; purpura hæmorrhagica, one; secondary post-partum hæmorrhage, two; myeloid leukæmia, one; subacute bacterial endocarditis, one; perforated iliac artery in efforts to cause abortion, one; hæmolytic anæmia, one; ruptured cerebral aneurysm, two; incarcerated fibroid of the uterus at the third month of pregnancy, hysterectomy, pulmonary embolus six days after operation, one; thrombophlebitis, one; acute poliomyelitis, two; chronic nephritis, one; reticulosarcoma, one; acute glomerular nephritis in puerperium, one; ruptured uterus at 39 weeks due to an epileptic fit, one; pulmonary tuberculosis, one; meningocarcinomatosis, one; chronic pyelonephritis and renal failure, one; cancer of lung, one; acute porphyria, one; generalized carcinomatosis, one; encephalitis, one; cerebral tumour, two; ruptured appendix on third day of puerperium, one; intestinal obstruction, one; contracted pelvis, obstructed labour, forceps application, death under anæsthesia, one; influenza and pneumonia, one; diabetes, two; acute yellow atrophy of the liver, one; unknown cause, two.

As has previously been stated in the section on toxæmia, deaths in which either chronic hypertension or chronic glomerular nephritis was the primary cause have been included as "maternal" deaths, though in the International Classification of Causes of Death, in common with all other diseases existing before pregnancy, they are classified as "non-maternal", irrespective of whether or not the disease may have been aggravated and death hastened by the supervention of the pregnancy.

Non-Maternal Deaths from Chronic Heart Disease.

Seventeen deaths from chronic heart disease were investigated by the Committee during the seven-year period covered by this report. Of these deaths, 11 were considered to have been preventible. The commonest form of heart disease encountered was mitral stenosis (10 cases). The majority of patients were aged over 32 years, and were multiparæ with two or more children.

CASE 41.—This patient was a multipara, aged 38 years. She had had two or three full-time confinements, but nothing is known regarding the pregnancies. In this, her third or fourth pregnancy, she did not seek ante-natal care until she was 30 weeks pregnant. Her blood pressure was then 135/105 mm. of mercury and her urine was normal, but she was not weighed. She complained of "shortness of breath on exertion and slight swelling of the feet". Examination revealed "only moderate œdema of the ankles". The chest findings were normal and the liver was not enlarged, but there was a presystolic murmur in the mitral area and a thrill was present. The pulse was regular and normal. She had "old rheumatic mitral stenosis". The doctor advised her "to rest and see me in one month's time if feeling well, otherwise to see me before".

One month later, while lying on her bed at home, she complained to her husband that she did not feel well. She then rolled over "made a gurgling noise in her throat, and became unconscious". The doctor arrived within five minutes, but found her dead.

The cause of death as stated on the death certificate was: "I. (*a*) cerebral embolism; (*b*) mitral stenosis; II. early pregnancy." No post-mortem examination was performed.

Comment.—This case illustrates the need for admission of the patient to hospital for bed rest, freedom from domestic responsibilities, nursing care and medical supervision when there are even early signs of congestive failure in heart disease in pregnancy. It is generally useless to advise such a patient to rest at home and return in a month if well, and sooner if not so well, as was done in this case.

TABLE IV.

Deaths from Cæsarean Section, 1950 to 1956.

Identification.	Age. (Years.)	Parity.	Indication for Operation.	Cause of Maternal Death.	Child: Alive or Stillborn.	Time after Operation when Death Occurred.	Status of Operator.	Type of Anæsthetic.	Status of Anæsthetist.	Blood Ready in Theatre.	Blood Transfusion.
				"Maternal" Causes.							
Year 1950[1]:											
1 ..	38	4	Toxæmia: ruptured uterus.	Uræmia: ruptured uterus.	Alive.	6 days.	Consultant.	Oxygen, ether.	Resident medical officer.	Yes.	Given.
2 ..	41	5	Intestinal obstruction.	Toxæmia: shock.	Stillborn.	1 day.	Consultant.	"Pentothal", oxygen, nitrous oxide.	Honorary medical officer.	Yes.	Given.
3 ..	31	1	Disproportion: toxæmia.	Toxæmia: cardiac failure.	Alive.	14 days.	General practitioner.	"Pentothal", nitrous oxide, "Syncurine", ether.	General practitioner.	—	Not given.
4 ..	36	2	Eclampsia.	Eclampsia: cerebral hæmorrhage.	Alive.	2 days.	Consultant.	"Pentothal", cyclopropane, oxygen.	Specialist anæsthetist.	—	Not needed.
5 ..	42	0	Eclampsia.	Intestinal obstruction.	Alive.	10 days.	Consultant.	Nitrous oxide, ether, cyclopropane.	Specialist anæsthetist.	—	Not needed.
6 ..	29	0	Placenta prævia.	Cardiac failure.	Alive.	6 days.	Consultant.	"Pentothal", curare, cyclopropane, nitrous oxide.	Specialist anæsthetist.	—	Given.
Year 1951[1]:											
1 ..	26	0	Eclampsia.	Eclampsia.	Alive.	Within 1 day.	Consultant.	"Pentothal."	General practitioner.	—	Not needed.
Year 1952[1]:											
1 ..	24	1	Disproportion: failed artificial rupture of membranes.	Sepsis.	Alive.	7 days.	Consultant.	Not stated.	Resident medical officer.	No.	Given too late.
2 ..	26	0	Maternal congenital deformity.	Sepsis.	Alive.	33 days.	Consultant.	"Tubarine", cyclopropane, "Pentothal", ether.	Specialist anæsthetist.	—	Yes.
Year 1953[1]:											
1 ..	25	0	Diabetes.	Adrenal and pituitary hæmorrhage.	Alive.	27 hours.	Consultant.	"Pentothal", tubocurarine, cyclopropane, nitrous oxide.	Specialist anæsthetist.	Yes.	Given.
Year 1954[1]:											
1 ..	39	2	Disproportion.	Intestinal obstruction.	Alive.	11 days.	General practitioner.	"Open ether."	General practitioner.	—	Inadequate.
2 ..	24	0	Inertia: disproportion.	Liver failure.	Alive.	7 days.	General practitioner.	"Open ether."	General practitioner.	—	Not given.
3 ..	21	0	Toxæmia.	Pulmonary embolism: thrombophlebitis.	Alive.	21 days.	General practitioner.	Not stated.	General practitioner.	—	Not known.
Year 1955:											
1 ..	35	1	Obstructed labour.	Hæmorrhage.	Alive.	30 hours.	General practitioner.	Nitrous oxide, ether.	General practitioner.	No.	Inadequate.
2 ..	41	0	Toxæmia.	Cerebral thrombophlebitis.	Alive.	36 days.	Consultant.	"Pentothal", cyclopropane, ether.	Specialist anæsthetist.	No.	Not needed.
3 ..	44	2	Chronic nephritis.	Uræmia.	Alive.	63 days.	Consultant.	"Pentothal."	Specialist anæsthetist.	Yes.	Given.
4 ..	35	1	Obstructed labour: "failed forceps".	Pulmonary embolism.	Stillborn.	42 hours.	Consultant.	"Pentothal", ether.	Not stated.	Yes.	Given.
5 ..	36	1	Obstructed labour.	Sepsis.	Alive.	4 days.	Consultant.	"Pentothal", cyclopropane, ether.	Specialist anæsthetist.	Yes.	Given.
6 ..	33	0	Toxæmia: failed artificial rupture of membranes.	Post-operative hæmorrhage.	Alive.	5 hours.	Consultant.	"Pentothal", "open ether".	Specialist anæsthetist.	Yes.	Given, but inadequate.
7 ..	35	1	Toxæmia: previous vaginal repair with gross scarring.	Pulmonary embolism.	Alive.	11 days.	Consultant.	"Pentothal", cyclopropane.	Not stated.	—	Given.
8 ..	22	0	Toxæmia.	Acute yellow atrophy of the liver.	Not stated.	10 hours.	General practitioner.	Not stated.	General practitioner.	No.	Not given.
Year 1956:											
1 ..	32	0	Placenta prævia.	Afibrinogenæmia.	Alive.	14 hours.	Consultant.	"Pentothal", cyclopropane, ether.	General practitioner.	—	Given.
2 ..	35	8	Twins: toxæmia.	Toxæmia.	Alive.	Within 1 day.	General practitioner.	"Open ether."	General practitioner.	Yes.	Not given.
3 ..	28	3	Placenta prævia.	Pulmonary embolism.	Alive.	4·5 hours.	General practitioner.	Nitrous oxide, "Pentothal".	General practitioner.	Yes.	Given.
4 ..	33	0	Disproportion: failed artificial rupture of membranes.	Sepsis.	Alive.	7 days.	Consultant.	"Pentothal", "Flaxedil", nitrous oxide.	Resident medical officer.	Not known.	Not given.

[1] Metropolitan cases only investigated (1950-1954 inclusive).

TABLE IV.—*Continued.*

Deaths from Cæsarean Section, 1950 to 1956.—Continued.

Identification.	Age. (Years.)	Parity.	Indication for Operation.	Cause of Maternal Death.	Child: Alive or Stillborn.	Time after Operation when Death Occurred.	Status of Operator.	Type of Anæsthetic.	Status of Anæsthetist.	Blood Ready in Theatre.	Blood Transfusion.
"Maternal" Causes.—*Continued.*											
Year 1956.—*Continued.*											
6 ..	47	8	"Failed forceps"; disproportion.	Hæmorrhage.	Alive.	6 hours.	General practitioner.	Ether, "Pentothal", nitrous oxide.	General practitioner.	Yes.	Given.
7 ..	30	0	Prolonged labour: fœtal distress.	Shock: Hæmorrhage: incompatible blood.	Alive.	4 hours.	General practitioner.	"Pentothal", cyclopropane.	Medical superintendent.	Yes.	Given.
"Non-Maternal" Causes.											
Year 1955:											
1 ..	37	1	To investigate a condition of intestinal obstruction.	Volvulus: cardiac failure.	Alive.	2 days.	General practitioner.	Ethyl chloride, ether.	Not stated.	No.	Not given.
Year 1956:											
1 ..	18	1	Chronic nephritis.	Chronic nephritis and superimposed pyelonephritis.	Stillborn.	7 days.	Consultant.	Local.	Specialist anæsthetist.	Yes.	Given.
2 ..	24	0	Diabetes.	Cerebral thrombophlebitis.	Alive.	3 days.	General practitioner.	"Pentothal", nitrous oxide, ether.	General practitioner.	—	Not given.

CONCLUSIONS.

As was stated earlier, the Special Medical Committee Investigating Maternal Mortality was appointed as part of a Scheme for the Reduction of Maternal Mortality which was inaugurated in 1939, and the object of their deliberations has been to ascertain whether reasonable facilities exist for the welfare of the mother and her baby, to assist in the maintenance of a high standard of obstetric practice in New South Wales, and to advise the Minister for Health accordingly.

During the years under reference, the investigation of all maternal deaths by the Committee was extended from the metropolitan area of Sydney to the whole State, with the result that a lack of certain facilities was revealed in areas outside Sydney, and that these were provided by the Department of Public Health in accordance with recommendations by the Committee to the Minister.

Services Now Established under the Scheme for the Reduction of Maternal Mortality.

The following services have now been established under the Scheme.

1. A free consultant service during pregnancy and confinement for all mothers in New South Wales who cannot afford a consultant's fee. Every medical practitioner is provided with an "Emergency Obstetric Service" booklet, in which is set out a list of obstetric consultants available for consultation in the metropolitan area only, in the area 50 miles beyond, or anywhere else in the State. Provision is thus made for direct contact with any of these consultants, and varying fees are paid according to the mode of contact and the distance. For instance, a fee is paid to the consultant and long-distance telephone charges are repaid to the medical practitioner. Consultation fees also vary according to time spent away from practice.

2. Free transport of the patient. To overcome some of the difficulties and hardships due to distance from large medical centres and bad travelling conditions, the cost of air, road or rail travel is paid to those families by which the additional expense of bringing a mother to a medical centre cannot be met.

3. Mobile blood transfusion units. With the cooperation of the Red Cross Blood Transfusion Service, the Women's Hospital, Crown Street, the Royal Hospital for Women, the Royal Prince Alfred Hospital, the Royal North Shore Hospital and St. George Hospital, a medical practitioner can call a mobile unit any hour of the day or night in Sydney. The Royal Newcastle Hospital provides the same service to any medical practitioner within 100 miles of the hospital. These mobile units are called directly by the medical practitioner, who must at that time provide certain accurate items of information to the hospital concerned. These requirements are simple, and are set out in the "Emergency Obstetric Services" booklet. The Department pays transport costs when this is indicated, and an honorarium to the medical officer and to the nurse from the hospital concerned. This service is of the greatest importance in saving the lives of mothers and their babies, and the cooperation of these hospitals is of great value.

4. Ante-natal care. (i) Mothers' record cards. These cards are provided free of charge to all medical practitioners. The object of these cards is to provide a "constant" record for mothers to retain in their handbags, so that they will be encouraged to attend regularly on the date indicated, and to ensure that this record will be taken to the hospital on their admission at a time when the out-patient records are not available and the dotcor's records are likely to be in his office. (ii) Suburban pre-natal clinics. Ten pre-natal clinics are conducted by departmental medical officers at certain baby health centres in Sydney. These services are located at centres remote from public maternity hospitals, so that expectant mothers can receive adequate pre-natal care in their own districts. Each mother is booked at her own hospital, where her blood count,

blood grouping, investigation for the Rh factor and Wassermann test are completed. So close is the liaison with the hospitals concerned that these clinics are, in fact, local out-patient departments. Trained dietitians from the department visit these clinics and advise the mothers on their food problems, particularly in cases of toxæmia. The mothers attending these clinics are saved much fatigue in travelling, long hours of waiting, anxiety over the minding of other children, hospital out-patient fees, and expense, fares and food.

5. Free booklet. A free booklet, "Healthy Motherhood", is available to all obstetric hospitals and medical practitioners for the mothers under their care. This contains much valuable information which is important to the mother; for instance, at the request of the Committee, a special inset was made, advising the mother to avoid solid food after the onset of labour. It also gives information concerning hæmatological and other services.

6. Free hæmatological and bacteriological services. At the request of the Committee, facilities for blood grouping and Rh investigation, which have always been available in Sydney for mothers who could not afford the additional fee, were extended to the remainder of the State. This was arranged by the cooperation of the Hospitals Commission, the public maternity hospitals outside Sydney, and the Director of the Red Cross Blood Transfusion Service. Facilities for bacteriological investigation of pyrexia or infection are available at the Institute of Pathology, Lidcombe, to any medical practitioner whose patient cannot afford the additional fee.

7. Foreign languages translations. The Departmental booklets are not translated into foreign languages, but early effective and practical contact with New Australian expectant mothers and mothers of young babies has been devised by translating two sets of questions and three special diets. These are available in 17 languages.

8. Physiotherapy in obstetrics. A film in sound and colour on "Physiotherapy in Obstetrics" for the training of medical students, physiotherapists and nurses is available. This sets out the latest advice on antenatal exercises and relaxation therapy, with demonstrations by expectant mothers. Information concerning these exercises is available in the booklet "Healthy Motherhood".

ACKNOWLEDGEMENTS BY THE MEDICAL SECRETARY.

The assistance of the officers of the Bureau of Statistics and Economics, Sydney, the Registrar-General's Department and the District Registrars, the Justice Department and the District Coroners has been of great value throughout the years under reference in the collection of data and in making available special advice concerning the correlation of information.

The members of the Publications Sub-Committee, Professor F. J. Browne and Dr. T. H. Small, have given invaluable service in the publication of this report by their careful consideration of many groups of original case histories, their critical appraisal of extracts prepared from case histories, and their evaluation of many tabulations. Without their assistance the report could not have been published; special thanks are offered to them for the time made available for this work. A tribute is also paid to the valuable work done by Dr. Wendy Cook in the preparation of abstracts of case histories and other material.

To all the members of the Special Medical Committee Investigating Maternal Mortality appreciation is expressed for their interest in the material forwarded to them, for the lively debates at meetings, and for their assistance in the practical following up of recommendations made by the committee for improved obstetric care throughout the State.

To the Chairman, Dr. H. G. Wallace, Director-General of Public Health, I should like to offer special thanks for his consistent encouragement to me throughout, for his keen interest in the work of the Committee, and for his help in the implementation of recommendations made by it.

The above are in addition to the acknowledgements already made at the beginning of this report.

GRACE J. CUTHBERT BROWNE, F.R.C.O.G.,
Medical Secretary.

Reviews.

A Short Practice of Surgery. By Hamilton Bailey, F.R.C.S. (Eng.), F.A.C.S., F.I.C.S., F.R.S.E., and McNeill Love, M.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S., F.I.C.S., with chapters by John Charnley, F.R.C.S. (Eng.), William P. Cleland, M.R.C.P. (Lond.), F.R.C.S. (Eng.), and Geoffrey Knight, F.R.C.S. (Eng.); Eleventh Edition; 1959. London: H. K. Lewis & Company Limited. 9½" × 6¼", pp. 1401, with 1697 illustrations. Price: £4 4s. (English).

THE appearance of a new (eleventh) edition of Bailey and Love's well-known textbook of surgery makes it obvious that it is an extremely popular book. Some of the reasons for this become apparent as soon as it is handled, for its material has been arranged and presented in a manner which holds the reader's attention from the outset. The text itself is well arranged, the different print types being used most skilfully. The numerous illustrations, diagrams and tables are also attractively displayed.

Are these first good impressions maintained during a more thorough perusal and with everyday use of the book? The answer is an emphatic "Yes". The text is brief, but it is lucid, and all the necessary information is included. Short reviews of the relevant anatomy and physiology are given, and these incorporate most of the present-day ideas. In its clinical descriptions and in its pictorial illustrations the book excels. The various sections are well balanced, and in each the relative importance of the conditions described is made clear. The reader is, in fact, given an excellent over-all picture of modern surgical trends.

In a book which covers the whole field, there are many points in which disagreement with the authors will be felt, and a whole list of such points could be compiled here. However, this is primarily a student's manual, and it will give him a first-rate grounding. He would do well to inquire of his teachers why they disagree in their opinions on the various points. In most instances their reasons will have to be good ones.

The value of the book would be increased by the provision of a series of references to the literature. The biographical details included in the footnotes are very interesting; but surely it is more important to know where, when and how a particular writer said his piece rather than who he is. The acquisition of a list of names and dates is no substitute for a true study of surgical history and philosophy.

In short, this is an excellent, justifiably popular textbook, which can be wholeheartedly recommended. It might well be subtitled "Surgery Without Tears".

Parasitology (Protozoology and Helminthology) in Relation to Clinical Medicine. By K. D. Chatterjee, M.D.; Second Edition; 1959. Calcutta: K. D. Chatterjee. 9½" × 6¼", pp. 196, with 92 illustrations. Price: Rs. 17.50 (India).

THE second edition of this useful book comes to us printed on better quality paper with a few additions and improvements, while its modest dimensions are still maintained. Chief among the additions are 21 extra illustrations, including 10 new coloured plates. Of these, one showing the histological features of the spleen in kala-azar and two depicting the pre-erythrocytic stages of *Plasmodium falciparum* and *P. ovale* are the most noteworthy. These last named are reproduced from the classical papers of Shortt *et alii* (1951) and Garnham *et alii* (1955) respectively, in the *Transactions of the Royal Society of Tropical Medicine and Hygiene*. There appears to us to be less reason for reproducing in colour the enlarged scolices of *Tænia saginata* and *T. solium*.

Some inaccuracies that had crept into the first edition have been corrected in the present one, which, however, still contains some ambiguities and one or two statements with which all parasitologists would not agree. When it is stated that the sexual cycle of the malarial parasite occurs in the brain capillaries in cerebral malaria, the author is evidently referring to the production of gametocytes in this situation. This phenomenon was described many years ago by Gaskell and Millar, but is not a commonly occurring feature of *P. falciparum* infections.

Nor would protozoologists subscribe to the view that polymorphism in trypanosomes refers only to the flagellar characters (page 33).

In the section on the vectors of filariasis, an error has been made in naming *Aedes scutellaris* as a vector in the Pacific, when *A. pseudoscutellaris* was probably intended. The point is more than academic, since the type species is inhospitable to the parasite, whilst its near relatives *pseudoscutellous* and *polynesiensis* are highly hospitable.

The few defects mentioned are overwhelmingly outweighed by the many excellences of the book, which is again warmly recommended to students of human parasitology.

Treatment of Malignant Blood Diseases by Radioactive Phosphorus: Part II. Hematological Aspects. By Ingmar Bergström; Acta Radiologica, Supplement 159; 1957. Stockholm: Acta Radiologica. 9½" x 7", pp. 98, with 69 illustrations. Price: Sw. Kr. 25.

THIS is an exhaustively complete account of the changes occurring in blood and bone marrow during the treatment by P^{32} of "immature" leukæmia, chronic lymphatic leukæmia, chronic myeloid leukæmia, multiple myeloma and a variety of malignant lymphomas. There is nothing new or unexpected in the author's findings, and the text is profusely illustrated by crowded graphs, which chiefly serve to illustrate the natural variations of the disease.

Little consistent effect was seen in "immature" leukæmias; but in chronic myeloid and chronic lymphatic leukæmia, satisfactory reduction in the circulating cells was obtained without adverse effects on the normal circulating elements of the blood. In the terminal stages, the diseases were not controlled, and there was noticed throughout a progressive resistance of the malignant cells to therapy. The return of the marrow towards normality was partly gauged by the reappearance of fat cells in sections of marrow. There was usually some degree of persistent leukæmic infiltrate in both myeloid and lymphatic types. Long-continued therapy with P^{32} did not cause significant marrow fibrosis beyond what might be considered normal for the disease being treated. Multiple myeloma was little influenced by this form of therapy.

The Mast Cells. By James F. Riley, M.B., Ch.B. (Hons.), M.D., Ph.D., D.M.R.T., F.R.C.S.E., with a foreword by Sir Henry Dale, O.M., G.B.E., F.R.C.P., F.R.S.; 1959. Edinburgh and London: E. and S. Livingstone Limited. 9½" x 6½", pp. 194, with 65 illustrations. Price: 30s. (English).

IN 1953, following an idea dating back to his student days, James F. Riley was able to demonstrate that histamine liberators reacted with mast cells, and in collaboration with G. B. West, tissue concentrations of histamine were correlated with the mast-cell population. Since then there has been a good deal of work upon mast cells, of which the most important and the most interesting has been that of Riley and West. Consequently a book written about mast cells by Riley is most welcome.

The first part of the book is a review of the literature. Most of the book, however, is devoted to experimental studies upon the histamine-release phenomenon in various animals, but particularly in the rat. There is also a description of the distribution of mast cells, with an account of those conditions, including tumours, in which mast cells are found to be increased.

In general this book can be said to contain most of the known facts about mast cells. Nevertheless, the actual function of these cells remains unknown. No light is thrown upon the function of histamine, nor is the mechanism of anaphylaxis elucidated. Whereas anaphylactoid states can be induced in various animals by the use of histamine-release agents which cause the disruption of mast cells, the injection of histamine itself does not always reproduce those phenomena.

It seems fairly certain that mast cells contain heparin as well as histamine, yet it is only in the dog that the phenomenon of histamine release is followed by increased clotting time of the blood. Because of this, the author of this book has reverted to Ehrlich's original suggestion that mast cells play a part in the nutrition of connective tissues. Riley believes that the main function of heparin is not in its anticoagulant activity; he suggests, instead, that it is a precursor of hyaluronic acid.

Sir Henry Dale, himself a pupil of Paul Ehrlich, has supplied a foreword to this most informative monograph. No person interested in the fundamental reactions of tissues and organs can afford to ignore this book, for here is the background which must be understood before such mechanisms as those of inflammation and allergy, amongst others, can be unravelled.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Cranial Nerves: Anatomy and Anatomico-Clinical Correlations", by Alf Brodal, M.D.; 1959. Oxford: Blackwell Scientific Publications. 7¾" x 4¾", pp. 144, with 25 illustrations. Price: 15s. (Abroad).

"Malariology With Special Reference to Malaya", by A. A. Sandosham, L.M.S. (S'pore), Ph.D (Lond.), F.R.E.S., F.L.S., F.Z.S., F.R.M.S.; 1959. Singapore: University of Malaya Press. 8½" x 5½", pp. 348, with many illustrations. Price: 59s. 3d.

"The Year Book of Endocrinology (1958-1959 Year Book Series)", edited by Gilbert S. Gordan, M.D., Ph.D., F.A.C.P.; 1959. Chicago: The Year Book Publishers. Sydney: W. Ramsay (Surgical) Limited. 7¾" x 5", pp. 384, with 83 illustrations. Price: £4 2s. 6d.

"The Clonal Selection Theory of Acquired Immunity", by Sir Macfarlane Burnet, O.M., F.R.S.; 1959. Cambridge: University Press. 8½" x 5½", pp. 222. Price: 22s. 6d.

"The Year Book of Neurology, Psychiatry and Neurosurgery (1958-1959 Year Book Series)." "Neurology", edited by Roland P. Mackay, M.D., "Psychiatry", edited by S. Bernard Wortis, M.D., "Neurosurgery", edited by Oscar Sugar, M.D., 1959. Chicago: The Year Book Publishers. Sydney: W. Ramsay (Surgical) Limited. 7¾" x 5", pp. 624, with 107 illustrations. Price: £4 13s. 6d.

"Childbirth Without Pain", by Dr. Pierre Vellay and others; 1959. Translated from the French by Denise Lloyd. London: Hutchinson of London with George Allen & Unwin, Limited. 5½" x 9", pp. 216, with illustrations. Price: 35s. (English).

"Clinical Dermatology: For Students and Practitioners", by Harry M. Robinson, Jr., B.S., M.D., and Raymond C. V. Robinson, B.S., M.D., M.Sc. (Med.); 1959. Baltimore: The Williams & Wilkins Company. Sydney: Angus and Robertson, Limited. 10¾" x 7¾", pp. 258, with 116 illustrations. Price: 93s. 6d.

"Proceedings of the World Congress of Gastroenterology and the Fifty-ninth Annual Meeting of the American Gastroenterological Association: Washington, D.C., U.S.A., May 25th through 31st, 1958", Volumes 1 and 2; 1959. Baltimore: The Williams & Wilkins Company. Sydney: Angus and Robertson, Limited. 10" x 6¾", pp. 1364, with many illustrations. Price: £11.

"International Work in Bilharziasis, 1948-1958", World Health Organization; 1959. Geneva: World Health Organization. 9¾" x 7", pp. 58, with illustrations. Price: 1s. 9d. or Sw. Fr. 1.

"Pain: Problems Pertaining to the Transmission of Nerve Impulses which Give Rise to Pain; Preliminary Statement", by W. Noordenbos; 1959. Amsterdam, London, New York and Princeton: Elsevier Publishing Company. London: D. van Nostrand Company, Limited. 8¾" x 5½", pp. 192, with 39 illustrations. Price: 42s. 6d. (English).

"Clinical Reports of the Adelaide Children's Hospital", Vol. 3, No. 2; 1958-1959. Adelaide: The Adelaide Children's Hospital, Inc. 9¾" x 7¾", pp. 101-152, with many illustrations. Price not stated.

"Principles and Practice of Obstetric Anæsthesia", by J. Selwyn Crawford, M.B., Ch.B., D.A. (Eng.), F.F.A., F.C.S.; 1959. Oxford: Blackwell Scientific Publications, Limited. 8¾" x 5½", pp. 136, with illustrations. Price: 20s. (English).

"Introduction of Radiation Medicine into the Undergraduate Medical Curriculum", Fifth Report of the Expert Committee on Professional and Technical Education of Medical and Auxiliary Personnel. World Health Organization Technical Series, No. 155; 1958. Geneva: World Health Organization. 9¾" x 6¾", pp. 24. Price: 1s. 9d.

"The Life and Times of Sir Charles Hastings: Founder of the British Medical Association", by William H. McMenemey, M.A., D.M., F.R.C.P., D.P.M.; 1959. Edinburgh and London: E. & S. Livingstone, Limited. 8¾" x 6", pp. 528, with illustrations. Price: 50s.

"Survey of Clinical Pediatrics", by Lawrence B. Slobody, M.D.; Third Edition; 1959. New York, Toronto and London: McGraw Hill Book Company, Incorporated. 9" x 5¾", pp. 566, with 32 tables. Price: $11.00.

The Medical Journal of Australia

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OBSERVATION AND EXPERIMENT.

IN his Cutter Lectures,[1] a few years ago, Professor A. Bradford Hill discussed the value of statistics for systematizing and interpreting observations in medicine, a means of approach that is not as recent in origin as might be supposed. The first great classic of medical statistics was the commentary of John Graunt on the London Bills of Mortality written in 1662, and English authors and administrators have since made much use of statistics. We may instance the work of William Farr at the office of the Registrar-General, of John Snow as a medical officer of health and of Sir John Simon as a health legislator. Often, as in John Snow's work on cholera in London (1847), it was possible to take effective action even though the bacterial agent was unknown at the time, because empirical relations had been established by statistical methods between disease and environment, in Snow's case between cholera and the water supply. Although Pierre Louis had carried out statistical studies on typhoid and tuberculosis before this time, his work was not followed up by others. Louis was hampered because he did not have a well-developed body of statistical technique for testing hypotheses, and it would have needed very large samples, involving the clinical observations of many workers, to establish the reality of some of the differences he was interested in. Nor were the clinicians of his time well disposed towards any critical appraisal of current methods. Experimental methods, however, gradually became adopted in epidemiological and pharmacological work, and statistical methods were found to be essential for such operations as the titration of biological products, the testing of the efficacy of antisera in laboratory animals and so on. There remained a gap between the results in the laboratories and the effects of these procedures applied to human populations. Diphtheria immunization might be regarded as an example. This was effective in animal populations, but further evidence was needed before it could be proved to be of value in human populations.

In another place (the Harben Lectures)[2] Professor Hill cited the English epidemiologist, Sir John Ledingham, as saying in 1939 that "though our laboratory workers have contributed very materially to the knowledge of methods, we have left to other countries the task of trying them out seriously and intensively in the field". Hill goes on to stress both the importance of experimentation and the difficulties. The essence of good experimentation is planning, so that the maximum of information can be extracted from a given amount of effort. Treatments must be allocated and subjects used in such a way that specific questions raised before the experiment can be answered. Often, of course, experiments of a type that would be practical and desirable in veterinary or agricultural practice are forbidden by ethical considerations. As an example, high oxygen tension in the treatment of the premature child leads to retrolental fibroplasia, but no experiment to establish this more firmly is ethical. Likewise, in animals one can test the possibility that noxious stimuli lead to congenital abnormalities or that carcinogens produce cancers, but no controlled experiments of this kind can be even considered in the human subject. On the other hand, many clinical procedures can be regarded as experiments even though they fall well below the best standards. The routine removal of tonsils is a good example of such an uncontrolled experiment. This procedure is now under a good deal of criticism, and yet many millions of such operations have been performed. In the earlier years, some form of experiment in which comparable children were treated differently would have been invaluable, and probably it could have been arranged in such a way as not to have broken any ethical rules. But ethical considerations are not the only source of difficulties in medical experimentation. Some diseases are not common, or the morbidity rate may not be high. Such is the case with poliomyelitis, in which actual symptoms occur only in a small minority of those infected. Field trials then will need to include perhaps a million treated subjects to test the efficacy of a vaccine. Special organizational problems arise. Bradford Hill gives a number of references to the various field trials carried out by the Medical Research Council Committees, in which such problems have been met and overcome. But therapeutic trials are also necessary in clinical medicine, and English experience has shown that cooperation can be obtained if, first, the treatment to be tested is likely to be of value in the human subject, second, its efficacy in the human has not already been adequately tested and, third, the drug has relatively unimportant side effects. On the subject of volunteers, Hill makes some important points. Many field trials have been ruined by comparing the experience of volunteers for a vaccine with others who have not volunteered. It can often be shown that such comparisons are not valid—the volunteers being, perhaps, of more conscientious type or of different social class. Hill looks with disfavour on some definitions of volunteer—young babies who "volunteer" through their guardian, convicts who have been promised shorter terms, and victims of incurable disease—and notes that "to go so far as to hazard life and health in the human subject, however circumstanced, might be, I suggest, to hazard debasing the outlook of the experimenter itself". He stresses the importance of retaining the correct doctor-patient relationship and of not allowing deceit to creep into these relationships.

These two papers by Bradford Hill can be confidently recommended to all who are interested in the planning of medical research. To the examination of statistical

[1] *New Engl. J. Med.*, 1953, 248: 995.
[2] *J. roy. Inst. publ. Hlth*, 1958, 21: 177, 185.

method is added wise counsel on human experimentation. The importance of the latter cannot of course be overemphasized, and we should like to draw attention again to a valuable group of papers by M. B. Shimkin and others which appeared in the issue of *Science* for February 27, 1953, and on which we commented at the time.[3] The subject has also been very ably discussed by R. A. McCance.[4] Experiments or trials are possible, desirable and ethical in medical practice, but constant vigilance is required to see that undesirable features do not enter.

Current Comment.

UNEXPECTED SUDDEN DEATH IN YOUNG ADULTS.

EARLY in 1958 the Press reported the sudden death of a young woman in New South Wales from an unknown cause. The coroner's finding was apparently that she just ceased to exist. This has aroused interest and provoked inquiries from medical men in other parts of the world; so we are now publishing the details of the case so far as we have been able to obtain them.

A young woman, aged 17 years, who was employed in a retail store in a country town in New South Wales, rode her bicycle from the store to her home for her mid-day meal. She rode back and resumed work. About one hour later, whilst standing in the store, she was seen to slump sideways and fall to the floor. She was breathing heavily, and she apparently died quickly without speaking. It was stated that on this day she had been in her usual state of good health, and that she had suffered only from children's ailments. Evidence was given that she was seen riding her bicycle back to the store, that one part of the road had a fairly steep grade, and that it had been her custom to stand on the pedals when riding up this hill. An autopsy was performed, but even after microscopic examination of tissues no cause of death was found. The opinion given was that death was a natural one and that life suddenly ceased to exist. The coroner's finding was that the deceased collapsed and died from natural causes, but from what actual cause the evidence adduced did not enable him to say.

In this age of coronary artery disease, sudden death is not uncommon, and the pathologist usually has no difficulty in pointing to the cause. Sudden unexpected death in a young adult who had always seemed healthy is rather perturbing, and the perturbation is intensified when no cause of death has been found. Sudden unexpected death in young persons may be due to a variety of causes, such as vagal inhibition, endocardial fibroelastosis, myocarditis, various cardiomyopathies and, of course, coronary artery disease. In some cases, the heart may appear normal to one inexperienced in autopsy work, and one cannot help feeling that in the case quoted above, there was some demonstrable but unrecognized cardiac cause of death.

This brings to mind the interesting paper of Donald Teare,[1] who described nine cases of asymmetrical hypertrophy or muscular hamartoma of the heart. Eight of these caused sudden death in young adults. In most cases death occurred during or just after some exertion. There is also a suggestion of familial incidence, as in his Case 5, that of a young woman, aged 21 years, who collapsed while running for a bus and was dead by the time she was taken to hospital. In this case there had been a past history suggestive of cardiac disease, and the clinical diagnosis made some years before rested between Ebstein's syndrome and endocardial fibroelastosis. This young woman's brother, aged 16 years, collapsed and died while riding his bicycle. No previous medical history was available. At post-mortem examination he was found to have changes in his heart of localized hypertrophy affecting the anterior wall and interventricular septum which were virtually identical with those of his sister. By coincidence, on the day of his death his younger sister attended the out-patient department of Hammersmith Hospital and was found to have signs identical with those exhibited by her deceased sister. The hypertrophic area of heart in these cases consisted of irregular bundles of muscle arranged in such a way as to suggest the impossibility of coordinated contractions. The lesion is often localized to the interventricular septum, but it may encroach on the atrio-ventricular valves, giving the signs of mitral or tricuspid stenosis. One of Teare's cases actually underwent operation for "mitral stenosis". Although a rare condition, asymmetrical hypertrophy of the heart is important, since it is a cause of sudden death in young or relatively young people, and it may explain the sudden deaths of siblings which are occasionally publicized in the popular Press.

[3] *MED. J. AUST., 1953, 2:23 (July 4).*

[4] In "Medical Ethics", edited by M. Davidson, Lloyd-Luke, London, 1957.

[1] *Brit. Heart J., 1958, 20:1.*

THE MECHANISM OF MIGRAINE.

IT is generally believed that the aura of a migraine attack is associated with constriction of cerebral arteries, while excessive vasodilatation is responsible for the subsequent throbbing headache. In an annotation published in *The Lancet* earlier this year,[1] the vascular aspects of migraine attacks are discussed. It is suggested that parasympathetic overaction is a factor. Some work by E. C. Kunkle[2] on this aspect is quoted. Kunkle states that in five out of nine cases of "intracranial" headaches an acetylcholine-like substance was demonstrated in the cerebro-spinal fluid, while in none of ten cases of "extracranial" headaches was it found. The criteria for "intracranial" headaches are not beyond criticism. The annotation ends with the sentence: "Perhaps we shall have no answer to this until we learn more of ætiology—of what throws the migrainous mechanism into activity."

In a recent letter to the editor of *The Lancet*, J. A. Hamilton,[3] writing from Stanford University Medical School, discusses "what throws the migrainous mechanism into activity". He poses the question in another way: "What interferes with the homœostatic mechanism which maintains cerebral vessels midway between extreme vasoconstriction and excessive vasodilation?" Hamilton, in an extensive study on respiratory alkalosis in cases of anxiety, has shown that when anxiety is associated with hyperventilation the patient is confronted with an array of distressing symptoms secondary to carbon dioxide deficit. These can be interrupted almost immediately by inhalation of carbogen (oxygen with 5% carbon dioxide). A number of patients being available with classical migraine, the effect of inhaling carbogen was determined at the onset of the aura. Almost without exception the aura disappeared, and the expected cephalalgia did not materialize. The patients were then instructed to inhale carbogen for five minutes three times a day for varying periods. Recurrences of even the aura were rare, and when the regular inhalations ceased, migraine attacks usually failed to appear for months.

Does this help to explain the factors concerned in the occurrence of a migraine attack? To a certain extent it may. Hamilton comments that under non-pathological conditions cerebral and perhaps other vessels are maintained by homœostasis between mild and temporary vasodilatation. Certain factors, extraneous to the carotid tree, among them respiratory alkalosis, tend to induce a severe and continuing vasoconstriction. Finally the arteriolar muscles become exhausted and complete dilatation occurs, with stimulation of pain end-organs in the vessel walls. This still does not explain why respiratory alkalosis or some other cause of vasoconstriction occurs.

[1] Lancet, 1959, 1:720 (April 4).

[2] *Arch. Neurol. Psychiat.*, 1958, 81:135 (February).

[3] *Lancet, 1959, 2:412 (September 19).*

Still, the inhalation of carbon dioxide might well be tried in the management of severe migraine attacks, particularly in the period of aura.

THE ORIGIN OF LIFE ON EARTH.

THERE has been much speculation on the conditions on the earth at the time of the appearance of the first life. Many of our modern ideas are due to A. I. Oparin,[1] who pointed out that the spontaneous generation of the first living organism would have been more probable if large quantities of organic compounds had been present in the oceans of the primitive earth. Oparin further suggested that the primitive atmosphere would have been reducing, and that organic compounds might be synthesized under these conditions. The first organisms arising might be entirely dependent on the environment for organic substances. N. H. Horowitz[2] outlined how a simple organism might acquire the ability to perform various syntheses and eventually develop into an organism capable of synthesizing organic compounds from carbon dioxide, water and mineral salts. Other authors have failed to upset the hypothesis that a reasonable concentration of organic compounds could arise only in a reducing atmosphere. Heroic measures, such as the use of electric or ionic discharges from a cyclotron, have failed to obtain more than minute traces of organic materials such as formaldehyde.

H. C. Urey,[3] a chemist, has done much to develop our ideas of how the solar system may have been formed from cosmic dust clouds containing a great excess of hydrogen. The giant planets, Jupiter, Saturn, Uranus and Neptune, have still atmospheres of methane and ammonia, for with their lower temperature and greater mass the escape of hydrogen is more difficult than it is from the earth. It appears likely that the primitive earth had an atmosphere containing hydrogen, methane, nitrogen and ammonia and small amounts of the oxides of carbon. The change to the present atmosphere has been due to a progressive loss of hydrogen, which results in the production of nitrates, free oxygen and ferric compounds.

More recently S. L. Miller and H. C. Urey[4] have discussed in detail the escape of hydrogen, the equilibria of carbon and of nitrogen compounds, the synthesis of organic compounds, especially the synthetic activities of electric discharges occurring naturally, and the synthetic activity of ultraviolet light, radioactivity and cosmic rays. They point out that the older theories of the formation of the earth in a molten condition have largely been abandoned in favour of the view that it was formed from a cold cloud of cosmic dust. The earth's temperature rose, first, because of the release of gravitational energy and, secondly, because of radioactive energy. They believe that the temperature of the earth's surface probably never reached levels above 150° C., at which there would be degradation of any higher compounds formed. In other words, the conditions were such for a long time that organic compounds, once synthesized, would have a high chance of surviving for an indefinite period. Some workers have believed that the first organism may have been a polynucleotide capable of self-duplication. Oparin instead favours some colloid which was able to accumulate proteins or other substances from the environment, grow in size and then split into fragments, which repeat the process. Ultimately the coacervate (or conglomerate) would presumably begin to duplicate itself more efficiently and develop some sort of genetic apparatus. It must be conceded that this part of the story is highly speculative, but it seems that the discussion of Miller and Urey enables some hypotheses, such as the formation of organic materials in an oxidizing atmosphere, to be discarded. It is of interest that the authors believe that conditions may have been propitious for the development of life on Mars, Earth and Venus alone among the planets. They regard the search for evidence of life on Mars and Venus as one of the most important aspects of space travel.

[1] "The Origin of Life", 1938, Macmillan, New York.
[2] *Proc. Nat. Acad. Sci. U.S.*, *1945, 31:153.*
[3] "The Planets", 1952, Yale University Press, New Haven.
[4] *Science*, 1959, 130:245.

A GOITROGENIC FACTOR IN CRUCIFERA.

IN 1955 F. W. Clements[1] reported observations made in Tasmania which led to the formation of the hypothesis that a significant amount of the goitre in Tasmania was due to the action of an unknown substance in the milk of cows fed on chou-moellier, one of the Brassicæ. In 1957 Clements published a fuller account[2] of his observations on the subject. Members of the cabbage family have been implicated as causes of goitre for many years. In 1928 A. M. Chesney, T. A. Clawson and B. Webster[3] showed that feeding laboratory animals with cabbage would cause enlargement of the thyroid. In 1949 E. B. Astwood, M. A. Greer and M. Ettlinger[4] isolated from yellow turnips and Brassicæ seeds the active antithyroid substance 1-5-vinyl-2-thiooxazolidone, which they later called goitrin. Greer and his associates showed that goitrin did not exist in the plants as such, but as a glucosidal derivative of this substance termed progoitrin.

Greer and M. Deeney[5] now report a study of the antithyroid activity of goitrin and progoitrin in man. Early work showed that progoitrin was broken down to goitrin by an enzyme, myrosin, contained in crucifers, and studies indicated that cooking would usually destroy the potency of these plants, supposedly because of destruction of the enzyme. Thus only the ingestion of raw plants was thought to be likely to lead to the development of goitre.

Progoitrin has now been prepared in pure crystalline form, and Greer and Deeney have studied the action of progoitrin administered to man without the enzyme from the plant and have shown that progoitrin can be quantitatively converted to goitrin in the body by both man and rats. The antithyroid effect was determined by measuring the inhibition of radio-iodine uptake when radio-iodine was administered after the ingestion or injection of goitrin or progoitrin. Orally administered progoitrin was quite active in inhibiting the utilization of radio-iodine, and goitrin could be demonstrated in the blood and urine. Evidently the progoitrin is broken down in the intestine by an enzyme probably produced by bacteria, although this was not directly demonstrated. A reference is given by Greer and Deeney[6] to a paper by M. Kruela and M. Kiesvaara[6] in which the finding of goitrin in milk is noted.

On the evidence submitted by Greer and Deeney most people eating cooked vegetables containing progoitrin should be just as likely to develop goitre as when eating them raw. Considerable variation in response to a given dose of progoitrin was found, but it is not clear whether this was due to individual variations in the thioglycosidase activity of their gastro-intestinal tracts or in the response of their glands to antithyroid compounds. In order to assess the clinical efficacy of the antithyroid activity of progoitrin, one gramme was administered daily to a patient with fairly severe recurrent Graves' disease. The reaction was very satisfactory. The effect of a comparable dose of propylthiouracil (300 mg.) was studied. The results were strictly comparable. Although only one patient was studied, it would seem that progoitrin might be useful as an effective antithyroid compound in the treatment of thyrotoxicosis, especially as it has a prolonged effect when given in large doses. The clear demonstration that a pure substance, progoitrin, isolated from members of the Brassicæ, has a profound antithyroid effect, and that the amount of this in different plants varies considerably, lends support to the suggestion by Clements that the ingestion of these plants by man, directly or in milk from cows fed on these plants, can account for "epidemics" of goitre in various districts.

[1] MED. J. AUST., 1955, 2:369.
[2] *Ibid.*, 1957, 2:645.
[3] *Bull. Johns Hopk. Hosp.*, 1928, 43:261.
[4] *J. Biol. Chem.*, *1949, 181:121.*
[5] J. clin. Invest., 1959, 38:1465.
[6] *Acta chem. scand.*, 1958, 12:580.

Abstracts from Medical Literature.

PHYSIOLOGY.

Local Vasodilator Action of Carbon Dioxide.

A. DILL (*J. appl. Physiol.*, May, 1959) reports that immersion of the hand in water saturated with carbon dioxide causes an increase of about 40% in the rate of heat elimination to water at 29° C. This indicates a local vasodilatation of the resistance blood vessels.

The Control of Venous Return and Cardiac Output.

A. C. GUYTON *et alii* (*Amer. J. Physiol.*, May, 1959) consider the relative importance of venous and arterial resistances in controlling venous return and cardiac output. They report that in dogs with cardio-vascular reflexes completely blocked by total spinal anæsthesia the total peripheral resistance was increased fivefold or more in two ways: first, by injecting small plastic microspheres into the arteries, thereby increasing the arterial resistance, and secondly, by inflating pneumatic cuffs around the major veins, thereby increasing venous resistance. A small increase in venous resistance decreased cardiac output eight times as much as an increase in arterial resistance of similar magnitude. This difference was caused principally by (*a*) a marked rise in systemic arterial pressure when arterial resistance was increased, which maintained the cardiac output at almost normal levels, and (*b*) a fall in systemic arterial pressure when venous resistance was increased; this promoted an even greater fall in cardiac output than increased total peripheral resistance alone would have caused.

Maximal Heart Rate during Work in Older Men.

J. ÅSTRAND, I. P-O. ÅUSTRAND AND K. RODAHL (*J. appl. Physiol.*, July, 1959) report that nine male subjects, aged 56 to 68 years, performed muscular work up to maximum loads on a bicycle ergometer while breathing both ambient air and oxygen. Heart rate increased to an average maximum of 163 beats per minute. The maximum oxygen intake averaged 2·24 litres per minute and the blood lactic acid concentration 85 mg. per 100 ml. In no case was the maximum heart rate higher when breathing oxygen than when breathing air. This low maximum heart rate in older people probably limits the capacity for oxygen intake. Four subjects were able to work for about one hour without any sign of exhaustion on a work load requiring an oxygen consumption of about 50% of their maximal aerobic work capacity.

Vital Capacity and Maximum Breathing Capacity of Athletes and Non-Athletes.

D. G. STUART AND W. D. COLLINGS (*J. appl. Physiol.*, July, 1959) report that the vital capacity (VC), maximum breathing capacity (MBC) and MBC/VC measurements of 20 athletes and 20 non-athletes were compared. The mean VC score of the athletes was significantly higher than the mean VC of the non-athletes, but insignificant differences existed between the two groups in MBC and MBC/VC. It is suggested that the difference in VC is due to increased development of respiratory musculature incidental to regular physical training. This increase is not reflected in the MBC since this measurement would appear to be more concerned with the presence or absence of obstructive ventilatory defects that are unaffected by physical training. Results are compared with data from the literature.

Carbon Dioxide Gas Introduced into Coronary Arteries.

M. J. OPPENHEIMER *et alii* (*Amer. J. Physiol.*, June, 1959) report that carbon dioxide gas is well tolerated when introduced directly into coronary arteries of anæsthetized dogs. There were no fatalities among dogs with either normal or freshly infarcted hearts. Intra-coronary carbon dioxide gas produced no persisting changes in the electrocardiogram or in blood pressure when injected slowly. Rapid injections under pressure produced extrasystoles at the time of injection and caused some subsequent changes of short duration in the electrocardiogram. These short duration changes were alterations of *S-T* segment deviations (which had been purposely produced in the previous control period) toward a more normal configuration. During this same period of time coupled extrasystoles produced in the control period were suppressed.

Sodium Chloride Aversion of Hypertensive Rats.

M. J. FREGLY (*Amer. J. Physiol.*, June, 1959) reports that rats made hypertensive by encapsulation of both kidneys with latex envelopes manifest a relative sodium chloride aversion if given a choice between water and 0·15 molar sodium chloride solution to drink. The specificity of this aversion was tested by offering hypertensive rats a choice between salt solutions other than sodium chloride, and water. It was observed that hypertensive rats manifested aversions for potassium chloride, sodium sulphate, lithium chloride and sodium saccharin which were similar in character to the sodium chloride aversion. As soon as hypertensive rats were able to differentiate between any of these salt solutions and distilled water offered simultaneously, they rejected the salt solutions in favour of water. These animals never ingested more of any salt solution used than of distilled water. With the exception of the sodium saccharin solutions, normal rats ingested more salt solution than water and rejected salt solution only at concentrations three to ten times above preference threshold levels. Hence, it would appear that the sodium chloride aversion manifested by hypertensive rats is not specific, but part of a general salt aversion.

Reanimation of Mice Cooled to Less than 1° C.

J. A. MILLER, JR., AND F. S. MILLER (*Amer. J. Physiol.*, June, 1959) discuss the factors contributing to the successful reanimation of mice cooled to less than 1° C. They found that such mice can be reanimated if first cooled slowly to less than 20° C. in a sealed vessel of appropriate size. During this period the oxygen content of the vessel falls to 6%, the carbon dioxide increases to 11%, and the humidity to saturation. An analysis shows that hypercapnia is chiefly responsible for the success of the method, though hypoxia and increased humidity also are beneficial. This holds whether the percentage of recoveries, time and temperature of recovery of postural reflexes, or incidence of hind leg weakness is used as an index of success. During resuscitation the animals received artificial respiration with 100% oxygen, 95% oxygen plus 5% carbon dioxide, 90% oxygen plus 10% carbon dioxide, or air. Comparisons of percentage of recoveries, long-term survivals and uninjured hind legs show that 95% oxygen plus 5% carbon dioxide gives the best results, and 100% oxygen the poorest. These findings demonstrate the benefits of carbon dioxide in deep hypothermia.

Plasma Volume Changes Accompanying Transfusion Reactions.

J. W. REMINGTON AND C. H. BAKER (*Amer. J. Physiol.*, July, 1959) report that splenectomized dogs were infused serially with 5 to 10 ml. per kilogram of blood or plasma. There was a high incidence of reactions to the homologous plasma or blood, marked by a fall in arterial pressure, urticaria, cutaneous œdema, a prolonged bleeding time, laboured respiration and some fatalities. The pressure fall was not seen in anæsthetized dogs. The urticaria and skin œdema were largely prevented by injections of antihistaminic drugs, but the other reactions were not reduced. In the majority of dogs given homologous blood, the hæmatocrit showed a steep rise, indicating a disappearance of plasma. The reduced plasma volume was confirmed by T-1824 injections, although the agreement between hæmatocrit change and plasma volume change determined by the dye was not always quantitative. These changes were not clearly affected by the antihistaminic drugs. However, when dogs were given their own (autologous) plasma, the blood volume increase, as measured by dye or calculated from hæmatocrit change, was the same as that expected from the known amount of plasma transfused, corrected for ultrafiltrate shifts. The infusion reactions were also greatly curtailed.

Ventilation of Normal and Diseased Lungs.

W. A. BRISCOE AND A. COURNAND (*J. appl. Physiol.*, May, 1959) have studied the uneven ventilation of the lungs of six normal, four emphysematous and four other subjects by an open-circuit method, using 11% helium in air as the indicator gas. The volume and ventilation of two groups of alveoli, one more and one less ventilated, were assessed in most cases. A new graphic method of dealing with open-circuit mixing data is presented. The results are discussed and compared with those of other workers. Normal lungs can be said to behave as if about half their volume

was half as well ventilated as the rest of the lung. Emphysematous lungs can be said to behave as if three-quarters of the lung was only about one-fifth to one-tenth as well ventilated as the remaining quarter.

THERAPEUTICS.

Asthma in Children.

J. P. McGOVERN (*J. Amer. med. Ass.*, January 3, 1958) describes the treatment of asthma in infants and children. He states that the anxiety of the child and the parents is a strong factor in perpetuating the complaint, and that recurrent infections, nutritional problems and allergy all play a part. He recommends early treatment to dilate the bronchi with one-eighth to one-quarter of a grain of ephedrine and 15 mg. secobarbital per teaspoonful in a palatable suspension, which may be repeated in four hours. This may abort an attack of asthma. If the attack is severe, 0·1 to 0·4 ml. of one in 7000 aqueous solution of adrenaline should be given. To keep the tracheo-bronchial secretions loose, fluid is the best expectorant, because children readily become dehydrated. Small amounts of fluid of medium temperature should be given frequently, especially if there is vomiting. In severe attacks injection of adrenaline is advised rather than inhalation, because the latter tends to be used too much and induces a habit, and the inhalation then ceases to be effective. If bacterial infection is present, treatment with sulphonamides or a broad spectrum antibiotic is advised. Penicillin is not recommended on account of the tendency to severe reactions, and fatalities in allergic children. If the child is not better after such measures in 48 hours, 10 to 40 units of ACTH are given, followed by adrenal cortical steroids in a suitable dose, which is decreased over a five-day period to a maintenance dose for another five days. Steroids are then withdrawn and ACTH is given the next day to stimulate the adrenal cortex. If these measures fail, admission to hospital is advised, fluids are given parenterally, and oxygen, carbon dioxide and mist by the tracheo-bronchial route. Expectorant drugs such as potassium iodide or syrup of hydriodic acid may help. Inhalation of steam and a 5% to 10% concentration of carbon dioxide is adequate and well tolerated if administered at intervals. This has an expectorant effect, whereas oxygen has the opposite effect and, if given, should be combined with steam and carbon dioxide.

Paralysis Agitans.

L. J. DOSHAY AND K. CONSTABLE (*J. Amer. med. Ass.*, May 2, 1959) report the results of treatment of paralysis agitans with a new drug, chlorphenoxamine. This drug is a derivative of diphenhydramine hydrochloride ("Benadryl"). It is supplied in 100 mg. capsules and 50 mg. coated tablets. One hundred and sixty-one patients of the post-encephalitic, arteriosclerotic and idiopathic types were treated. The optimum dosage for most patients was 50 to 100 mg. thrice daily after meals, taken with milk because a few patients complained of indigestion when it was taken with water. Good results were obtained in 53% of patients in combating rigidity, fatigue, depression and weakness, but tremor was not relieved to the same extent. When the drug was used alone side reactions were very slight; it was also used on combination with trihexyphenidyl ("Artane"), ethopropazine or diphenhydramine with relief of tremor. Some patients ceased taking it on account of indigestion, but others have taken it for several months with benefit.

Chlorpromazine.

F. J. AYD (*J. Amer. med. Ass.*, March 21, 1959) records the effect of prolonged intake of chlorpromazine in 50 patients. Thirty-one women and 19 men aged 12 to 70 years were studied. The conditions for which the patients were treated were psychiatric ailments of many kinds, including anxiety states and obsessive compulsive neuroses. The doses of chlorpromazine varied between 100 and 2000 mg. daily. These patients took the drug for periods from two to four years. All patients gained weight. Amenorrhœa occurred in two-fifths of the women, but sometimes menstruation recurred with or without remission of the drug. No evidence of renal or hepatic dysfunction was found. In some cases the white cell count fell as low as 3000 per c.mm., but it was not necessary to restrict the drug as the count returned to normal while the chlorpromazine was continued. Three patients complained of photosensitization from May to October, but were desensitized by frequent limited exposures to the sun. The total dosage of chlorpromazine varied between 54,000 and 1,078,000 mg. None of the patients was cured, but many have returned to work or resumed the responsibility of the home, or household work. When an effort was made to withdraw the drug, symptoms almost invariably occurred. This study demonstrates that chlorpromazine may be used with benefit and without serious risk over long periods.

Hypertension Treated with Syrosingopine.

G. R. HERRMANN *et alii* (*J. Amer. med. Ass.*, April 4, 1959) discuss the treatment of hypertensive patients with syrosingopine administered orally. They state that rauwolfia and its derivatives are effective in many cases in reducing blood pressure, alone or in combination with other drugs, but that side effects such as drowsiness, nightmares, depression, nasal congestion, cramps, gastro-intestinal bleeding, fatigue, weakness, dysuria and impotence necessitate withdrawal in some cases. Syrosingopine (carbethoxysyringoyl methyl reserpate) is a synthetic compound derived from reserpine, which was given orally to 77 patients. Thirty-eight patients previously untreated were given 0·25 mg. of the drug four times daily, increased in some cases gradually up to 1 mg. four times daily. The blood pressure fell considerably in 16 patients, especially in those with a diastolic pressure above 120 mm. of mercury. Thirty-four patients on reserpine were transferred to syrosingopine (1 to 4 mg. per day) with slight decrease in blood pressure in some and increase in others. In another group of patients intolerant of rauwolfia preparations or of combined therapy with rauwolfia, chlorothiazide, hydrallazine and ganglion-blocking agents, syrosingopine in doses of 3 mg. per day was as effective as the previous regimen and without significant side effects. In three patients nightmares, nasal congestion and abdominal cramps persisted while they were taking syrosingopine, but in one case disappeared when the dose was reduced from 2 mg. to 0·5 mg. per day. The dose of syrosingopine used was larger than the dose of reserpine, but the side effects were less.

Flumethiazide, a Diuretic Agent.

A. C. MONTERO, J. B. ROCHELLE AND R. V. FORD (*Amer. Heart J.*, April, 1959) discuss a new diuretic agent, flumethiazide, which resembles chlorothiazide. Chemically, the only difference is a trifluoromethyl group instead of a chloride group. Pharmacologically, flumethiazide does not significantly differ from chlorothiazide in its diuretic potency or in the biochemical changes induced. The drug is effective used repetitively day by day. It is of potential value in the long-term management of various states of œdema and as an adjunct in the therapy of hypertension.

Imipramine.

J. DELAY, P. DENIKER AND T. LEMPRIÈRE (*Presse méd.*, May 9, 1959) have used imipramine in the treatment of 137 patients suffering from depressive and melancholic states. From their experience they conclude that imipramine therapy is at present the best form of chemotherapy for depressive states, which up to now have responded relatively slightly to medical treatment. The effects were excellent or good as a rule in more than 50% of cases. The greatest proportion of good results was obtained in endogenous, melancholic, presenile and organic depressive states, in which hitherto electroshock treatment has been indicated. The action of the new drug resembles that of electroshock by its radical modifying effect on the depressive outlook. As its effect appears less quickly and is less powerful, the indications for its use are slightly different. It may be used first in cases of light or medium severity. Moreover, since a prolonged course of the drug can be given, it is of value in relapses or protracted cases. Combination of the drug with the neuroleptics, especially levomepromazine, seems to increase its effect. It is easy to administer, and complications are uncommon; however, side-effects frequently occur. These are observed most often when large doses are given, or when the patients are predisposed to them by their age or by nervous damage. The principal action of the drug is as an anti-depressive and thymic stimulator; thus it may be classed among the psycho-analeptics or psychic stimulants. By its effectiveness, ease of administration and essentially interrupting action, imipramine seems likely to be used widely and for long periods. For this reason, the dangers that may accompany its administration by doctors who are not trained psychiatrists must be emphasized.

Points of View.

THE FUTURE OF THE AUSTRALIAN ABORIGINAL.

PROBABLY every one is agreed that the only way to prevent the complete disappearance of our aborigines is for them to pass into the general population and live as does the white population—to disappear just as the occasional Negro, Afghan and Chinese and individuals of other nationalities do in our midst. Is this possible? If so, how can it be brought about?

Twenty thousand years or more of separation of our natives from the rest of mankind has brought about changes in skin colour, in general physical features and, in my opinion, in their mental endowment. This latter is of a high order, but yet not quite the same as in Europeans. Nature—that is, inheritance—is the principal factor in our mental equipment. Nurture—that is, upbringing and education—plays upon the material that Nature has provided. Considering that his brain is that of a first-class nomad, it is not surprising that in the full-blood from time to time the "Call of the Wild" is a compelling force. Experience has shown that the aborigines are admirably fitted for station life dealing with cattle, horses and sheep, though there may be a little doubt as to their "stickability" in any particular job. In Europeans, the engineering type of brain must have existed in our primitive ancestors long before there was any possibility of its being applied along modern lines. There is reason to think that our natives have much latent mechanical ingenuity that may find expression in looking after farm machinery and station garages. In my opinion, therefore, our full-blood natives can take their place in the general pastoral community with reasonable success.

If this is possible, how can this assimilation be brought about? Attention may be called to the following points which have a bearing on this problem.

1. "In South Australia the aborigine possesses citizenship rights including the franchise although there are certain restrictive clauses provided in the Aborigines and Licensing Acts. On the other hand natives are entitled to many privileges not available to others vide Aborignes Act, Crown Lands Act, Fisheries Act, Animal and Birds Protection Act, Dogs Act, etc. . . . . In short aborigines who live a normal useful life can enjoy all the privileges of citizenship plus certain other privileges not available to other than aborigines."

2. In South Australia, under the *Licensing Act*, unless he is exempted, a full-blood native or half-caste cannot be supplied with alcoholic liquors. This provision was inserted because natives under the influence of alcohol may be exceedingly dangerous to the police and to white and other native citizens. An occasional murder still occurs from this cause; but presumably such crimes and the infliction of grievous bodily harm and rape are decidedly reduced by this provision. It is a pity that there is no ready way of branding a European who cannot take his liquor, so that he could be readily recognized and placed under similar prohibition. A native who is of good repute and of the required standard of intelligence and character can obtain an exemption, when he can, if he so desires, partake of intoxicants. As a help to him in withstanding the importunities of his kindred and friends, a provision is sometimes inserted on the certificate that the liquor supplied to him must be consumed on the licensed premises and not carried away to treat friends. Can anyone see any fault in these, to my mind, wise clauses?

3. Those of our natives who have white ancestors should be called "part whites" and not "part natives", so as to bring them more closely into the white fraternity into which they must ultimately merge. Several persons who have made good in various ways and are applauded as natives have a white ancestry, and it is difficult to know whether their ability is derived from the white ancestry or from able aboriginal ancestry or from both. Are there any examples of full-blood natives really successful in the higher walks in our social community?

4. In the first cross between an aboriginal and a European, the offspring have an equal number of genes from each parent. Such full half-castes intermarrying can produce children with varying numbers of genes from each side. Theoretically, of course, two full half-castes marrying could have one child with genes all white (and so European) or one with them all native (and so a pure-blood aboriginal), or with any degree of intermixture. It is scientifically absurd for Commonwealth authorities (or others) to say of any particular descendant of such marriages that aboriginal or European blood predominates.

5. In spite of the expenditure of very large sums of money and with the best of intentions, the Commonwealth Government cannot be said to have been more successful in its treatment of the natives than Western Australia, Queensland or South Australia. Victoria and New South Wales hardly come into the picture with their sophisticated part-whites, who are now a social problem rather than a native one. The three States first mentioned and the Commonwealth are all tackling the problem in somewhat different ways, with some success now in this and now in that direction. With four authorities thus engaged, each profiting by the experience of the others, progress is likely to be made. If the Commonwealth took over the whole control, the administration would be cast in a particular mould which might or might not be the best one, and experience would be reduced threefold. The people of Australia as a whole should, of course, pay the expense of the native situation; but it might be disastrous to a satisfactory solution if in paying the piper the Commonwealth insisted on calling the only tune that could be played.

6. In the *Police Act* in South Australia, until recently, there was a clause under which a person could be prosecuted for habitually consorting with natives without reasonable excuse. The term "consorting" in the *Police Act* has, of course, a sinister meaning. Unless married to native women, it is obviously undesirable that white men of loose morals should be allowed to cohabit with them. This is an offence under the *Aborigines Act;* but it is extremely difficult to catch such a rascal *in flagrante delicto.* To enable the situation to be controlled, especially in the far interior, where undesirable diggers and swagmen may take native women as temporary mistresses, this clause was inserted. If a white man was found living with natives, in a position to supply them with alcohol or to cohabit with their women or to exploit them for some financial gain, he could be prosecuted under this provision, unless he could show, as an honest man easily could, that his behaviour was beyond reproach. Even saints have succumbed to temptation and should be led away from it. The movement calling for the repeal of this clause was accompanied by unwise propaganda, misleading statements and misapplied sympathy.

7. The genes that govern mental endowment, as well as those responsible for bodily development, probably change slowly over hundreds of generations (producing the different races of man), and occasionally suddenly by a mutation. Native genes can be diluted by marriages with Europeans and so eventually become negligible. Full-blood natives, marrying amongst themselves and living under European conditions and receiving English education, would still have practically the same kind of mental and bodily genes after a number of generations. In other words, the native genes cannot be altered by European influences. One cannot say that it will take three generations or six to breed out the inherited native qualities. They will persist. Nurture, however, can exercise its influence on the inherited qualities, and this can be done in one generation. Take a full-blood native baby, bring it up from early infancy with white children and away from squalid surroundings, and the resulting adult should fit into the general community as reasonably as most people do, save for the important matter of colour, which may give rise to an inferiority complex.

8. The various Governments concerned are doing all that can reasonably be expected to fit our natives for assimilation. Educational facilities and medical attention are provided, officers of the Aborigines Department find employment for them, see that the aged and the sick are looked after, try to solve domestic difficulties and keep the young people from getting into trouble, and so on. It is up to the white population to treat the native people in friendly fashion, making them feel at one with the rest in community social activities. In the north, where a colour bar operates, station owners should see that the natives on their property—which is also, it must be remembered, in a sense the natives' property—are gradually induced to live in the European way in houses and to dress decently, the children being educated like white children.

9. The various Acts dealing with aborigines have all been passed with the objective of helping and, if necessary, protecting our natives. Every clause has been considered carefully before insertion. The intentions have been good. Attacks on the good faith of these Acts are much to be deprecated, and are used as capital for attacking the British Commonwealth by those who bear it ill-will; after all, is

there any other nation that can compare with Britain in its help to less fortunate races?

10. It is also a matter of deep regret that atrocities committed by unscrupulous white people on our natives many years ago are raked up and recounted. They are presumably exhumed for propaganda purposes. Greater atrocities are still being perpetrated even today by civilized communities.

11. The critics of administration could help very materially in the advancement of our natives were they prepared to meet them and help them individually, and particularly to undertake the upbringing of native children in European surroundings or even to adopt them. Such foster parents have, of course, to be approved as suitable to give the care required.

12. It may be said without equivocation that the solution of the native problem lies with the people of Australia generally, who must receive these people cordially and help them in their assimilation. The State authorities are doing all they should do; but it would not be in the interests of the natives for them to become the idle protégés of a welfare State.

13. Now that infanticide, starvation and disease are no longer taking any appreciable toll of native infant life, the full-bloods on the verge of civilization are multiplying at a considerable rate. In the Musgrave Ranges 114 children, counted in a dogging camp several years ago, must now in many instances be nearly grown up. The provision of food for this growing population must soon be beyond the capacity of the country. More important still is employment for these people, and employment would mean a food supply. Some can be trained as stockmen and station hands, but what of the others? Who can suggest other work that will appeal to the descendants of nomads and so supply their daily bread?

14. Some of our detribalized full-blood natives, camped in the outskirts of remote country towns, make wurlies for themselves out of any material available. Usually these are more efficient in protecting them from the wind and occasional rain than the boughs and porcupine grass of their ancestors. But, though such wurlies are draught-free and very cosy with a fire on the ground in the centre, to European eyes they are appalling with their old bags, flattened pieces of galvanized iron and so on. I can assure anyone that they are much more comfortable from the native point of view than a tin shed with an earth floor. No wonder that, when such accommodation is provided, the native is often found sleeping outside instead of in his European-provided shelter, which has even been known to be used as a lavatory. The casual visitor and the fault-seeking critic see much amiss in these wurlies, which may form the subject for propaganda to blame the authorities, when what is wanted is help from the local white people to enable the natives to reach a better standard of living.

15. There is frequent reference by well-meaning people to the granting of full citizenship rights to our natives. No one seems to have paused to consider what this term means. Except for recent Commonwealth legislation, which would not apply to South Australia, it has no legal meaning. With the exception of the franchise, a visitor, even an alien, and a minor enjoy the same protection as enrolled citizens, can buy and sell and sue and be sued as they do.

16. Our nomad natives in the interior are destitute of clothing. At night time, even the pubic tassle may be discarded. Warmth is obtained from small fires behind a breakwind. Obviously, the interposition of any clothing such as a shirt would cut off much of the radiant heat. One of the mistakes of the early days was to issue blankets to the wandering aborigines. The native, seeing the white man clothed, thinks that he presents a finer appearance if attired, however ragged and however incongruous the garment may be. When the natives used to visit the stations on the East-West line, they put on their cast-off clothing, not to excite sympathy, but because they considered that the passengers would be favourably impressed. Out hunting, they would wear nothing.

17. The aborigines in the older settled areas in South Australia are almost without exception "part whites". Very many of these have made good and have disappeared in the general white population. Those that remain in Government and mission stations are generally the more feckless. By the provision of houses in country towns where work is available, many of these have been induced to go out into the world, and most are holding their own.

18. A Governor-General of India once said, "Save me from zeal". A very wise remark. Don Quixote, with the best of intentions, tilted at wind-mills. There is no scandalous treatment of our natives. The authorities see well to this. But they do want a helping hand from the white people with whom they come in contact. They are an ancient race with many admirable qualities, much more like ourselves, except for colour, than the natives of Africa.

It is of interest to note that in "Bulletin No. 67 of the Royal Flying Doctor Service of Australia (South Australian Section)", dealing with trips from Alice Springs for the three months December, 1958, to February, 1959, 72 patients were concerned. Of the 72, 57 were natives and only 15 whites. The cost of the trips for the natives was nearly £300, borne by the administrations.

Adelaide. J. B. CLELAND.

British Medical Association.

THE HENRY SIMPSON NEWLAND PRIZE IN SURGERY.

THE Federal Council of the British Medical Association in Australia announces that the Henry Simpson Newland Prize in Surgery, established to commemorate the services of Sir Henry Newland to the medical profession, is open for competition. The conditions are as follows:

1. The prize shall be awarded every three years to the writer of the essay adjudged to be the best on a surgical subject.

2. The prize will consist of a money award of £100, together with a medal.

3. The next award will be made in 1962, the subject of the essay being "The Modern Management of Inflammatory and Neoplastic Diseases of the Left Colon and Rectum".

4. The dissertation should be based on personal observation and experience.

5. The essay must be typewritten or printed in English. It must be distinguished by a motto, and accompanied by a sealed envelope containing the name and address of the author and having on its outside the corresponding motto. It must not exceed 50,000 words.

6. Essays must be delivered to the General Secretary, Federal Council of the British Medical Association in Australia, 135 Macquarie Street, Sydney, not later than January 15, 1962.

7. The competition is open to any graduate of any medical school within the British Commonwealth.

8. The Committee administering the Henry Simpson Newland Prize Fund reserves the right to withhold the prize, and its decision in regard to any award shall be final.

9. The prize essay shall be submitted forthwith to the Editor of THE MEDICAL JOURNAL OF AUSTRALIA.

VICTORIAN BRANCH.

Essay on Asthma, 1960.

THE Alfred Hospital, through a gift from Miss Lillian Pratt, desires to encourage recent medical graduates to take an interest in asthma by offering two prizes of £25 each for suitable essays. It is proposed to offer one prize in 1960 and one in 1961.

The conditions are as follows:

1. The prize shall be awarded to the writer of the essay adjudged to be the best on a subject selected in 1960 and 1961.

2. The subject for 1960 is "The Management and Treatment of Bronchial Asthma".

3. The dissertation should be based on personal observation and experience of the writer.

4. The competition is open to medical graduates of not more than five years' standing, who are registered in any Australian State or Territory.

5. The examiners reserve the right to withhold the award.

6. Essays must be delivered to the Secretary, Section for the Study of Allergic Diseases, B.M.A. (Victorian Branch), c.o. 426 Albert Street, East Melbourne, C.2, Victoria, by August 31, 1960.

7. Each essay must be typewritten or printed and must not exceed 10,000 words in length.

8. Each essay must be distinguished by a motto, and must be accompanied by a sealed envelope marked by the same motto, containing the name and address of the author.

9. The examiners reserve the right to publish the prize essay.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

VACCINATION.

DR. WM. REDFERN TO LIEUT. GOVERNOR COLONEL PATERSON.[1]

[From the *Australasian Medical Gazette*, February 20, 1897.]

October 16, 1809.

Sir.

It is with extreme pleasure I at length feel myself enabled to state, with a degree of certainty, that my endeavours to establish the vaccine Inoculation with the VIRUS I had the honor of receiving from you, have perfectly succeeded. The reintroduction of so great a blessing to the rising generation, as an infallible, safe and mild Preventitive of one of the most fatal diseases to which the Human Species is liable, the SMALL POX: and which, fortunately for the Inhabitants of this Colony, has not yet made its appearance amongst them, will, I am confident, afford the most heartfelt satisfaction and highest gratification to your benevolent and philanthropic mind.

That the Communication was not made at an earlier period, I trust you, Sir, will not attribute to negligence or disinclination, as it was with the utmost pain and difficulty, I was able to carry on my experiments, from a very severe inflammation in my right hand, which commenced the very day after I had received the VIRUS and totally incapacitated me from writing: and, indeed, I was also unwilling to hazard a report of its success until I had established it beyond the possibility of doubt, which I am happy to say is now the case.

I have enclosed, Sir, for your information a list of those who have been VACCINATED, with the success attending it.

It now remains, Sir, for such measures to be adopted as your wisdom may suggest, that may appear best calculated to carry your benevolent intentions into effect, in order to diffuse it as generally as possible:—From those in the Superior Ranks of Life, we may, I presume, calculate upon every support that example and precept can furnish: but it becomes highly necessary to impress on the minds of the poorer orders of people, whose ignorance renders them but too susceptible of the grossest and most unfounded prejudices, the usefulness, safety and superior advantages of this new plan of Inoculation.

At the same time, I hope it will be managed with such judgment and discretion as will tend to keep it constantly alive: for there will always be a considerable risque of the VIRUS becoming effete from the length of time that must necessarily elapse in conveying it either from Europe or India. This object can only, in my humble opinion, be obtained by Inoculating but few at a time.

I remain, Sir,

With the greatest respect,

Your Honor's most obedient servant,

W. Redfern.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

THE CHALLENGE AHEAD.

SIR: I have been both amused and entertained by the excellent replies published in the Journal to the extraordinary ideas and theories of Dr. O'Day.

Surely a person who believes that (*a*) the Americans started the war in North Korea, (*b*) that the Russian atrocities in Hungary are due to imperialist counter-revolutionary elements supported by foreign encouragement and (*c*), worst of all, that the monstrous geneticide policy inflicted by the Chinese Communists on the hapless Tibetans is a good thing, will be gullible enough to believe anything, and cannot be taken seriously by thoughtful, intelligent people.

I would add one other point. Since it must by now be clear even to Dr. O'Day that the large majority of people in this country will have nothing to do with Communism or, for that matter, any other form of "ism", would he not be far happier living behind the iron curtain or in Communist China, where he can witness and even take part in the glorious Marxist experiment? He could also, no doubt, enjoy the spectacle of being surrounded by millions of political robots, who are forbidden under penalty of imprisonment, torture and death, from contemplating that unspeakable iniquity, all too common in this country, of actually criticizing their Government and political leaders.

Yours, etc.,

MEDICO-LEGAL.

Newcastle,
New South Wales.
December 11, 1959.

SIR: Dr. Sundin and Dr. Kater, in ridiculing Dr. O'Day's idealistic claim that Marxism is the non-idealistic science of politics, have not set him an example of scientific thought. Dr. Sundin is convinced that Marxists want to kill all who contradict their basic belief, Dr. Kater that Communism is based on killing everyone who does not obey. Both use war atrocities as proof of this. If this were so, Egyptians should believe, since the Suez incident, that most capitalists want to kill most Egyptians, as some British soldiers did atrocious things to some Egyptians.

Marx's "science" should be at least as fallible as Freud's or Lysenko's, considering that Marx deals with higher levels of complexity. This very complexity, however, should warn us not to fall into the same over-simplification trap, as Dr. O'Day has done. We will not effectively combat the theories of Marx, Freud or Lysenko by ill-directed and ill-informed abuse. Let us look at the motes in Marxist eyes by all means, squinting around the beams in our own as best we can, and see what excuses they give for their atrocities. Then let us see if the reasons we give for our military actions are much superior, if we switch the arguments from "our side" to "their side" and vice-versa. For instance, one Communist informed me that the first shots in the Korean war were fired, according to *The Sydney Morning Herald* of the time, at a point north of the Thirty-Eighth Parallel. Let us answer that factually before claiming that war saved Australia.

While with Dr. Kinsella I would not compare too closely Vatican policy with that of the Axis powers, I would not join him in equating Nazism with Communism, or dismiss them as evil philosophies, without knowing a great deal more than I do of *"Mein Kampf"* and *"Das Kapital"*; nor would I assume that atheism leads to totalitarianism, especially when I see some of the greatest atheists of our day fighting against totalitarian influences.

Hitler and Mussolini were professed theists, and Franco has been officially addressed as his church's "favourite son".

Yours, etc.,

D. N. EVERINGHAM.

P.O. Box 328,
Rockhampton,
Queensland.
December 14, 1959.

SIR: I do not desire to take part in the controversy in your today's number as regards Communism and Nazism, nor do I wish to express any political opinions; but as there is a tendency to throw both these systems into one basket, I would like to show that fundamentally they are entirely different, and in the German parliament the Nazis sat on the extreme right and the Communists on the extreme left to emphasize that in their whole philosophy and outlook on life they were as far apart as the poles.

The basic philosophy of Nazism, as it was explained to me, is built on racial hygiene, maintaining that not only the health but the upward development of mankind is

the sole aim in this existence, as nature's method of elimination and selection has ceased to function. It claims that all political government, economics, art and culture generally must serve this end. It maintains that all races and peoples, though they may each represent a supreme value in themselves, differ from one another and are marked off from one another by their own proper combination of bodily and mental characteristics which are transmitted from generation to generation by the laws of heredity, and that they are one and each developing in a direction which has been laid down in their past, and that the miscegenation of races and peoples too far apart in their genetic make-up will destroy the direction of each.

The carrying out of this philosophy is brought about by the education of children while still at school and the sterilization of the unfit, which, it is claimed, is being humanitarian as well as promotive, as thereby people are being prevented from coming into existence who can never enjoy a full and happy life. Religious worship is free for all denominations, and was reintroduced into the State schools after having been suspended by the Weimar Republic. Twenty-two of 24 States churches were united to form one Protestant State church, and a concordat was signed by the Vatican and the Nazi government. In economics private enterprise is maintained, but with considerable government interference to prevent exploitation. The political control is absolute and free of all influence by any form of money power. Roman law based on property was abolished, and old Aryan law introduced with emphasis on the protection of the human being. Although at the time of his passing Hitler was still governing under the old institutions, it was intended that the latter be abolished. Young men were to be picked for their suitability and trained as local and eventually county leaders, the supreme leader being eventually elected by a council of county leaders. A monarchy was provided for, to guard the constitution and refer the leader to the council if needed. Culturally and morally, all old values and ideals of the race were to be promoted and what was alien to be suppressed.

It seems to me that, looking back into history, all systems, whether for good or evil, wade through rivers of blood, and that it is useless to condemn on that account. The fault is not with the systems themselves, but unfortunately to have to admit, human nature itself.

Yours, etc.,

F. A. BÜTTNER.

131 Domain Road,
South Yarra, S.E.1,
Victoria.
December 12, 1959.

Notes and News.

The Orthoptic Council of New South Wales.

The Orthoptic Council of New South Wales announces that a course of instruction in orthoptics, of two years' duration, will commence on February 8, 1960. Applicants must be aged 18 years, or 17 years with their Leaving Certificate. They should apply to the Secretary, Dr. M. Sterling-Levis, 235 Macquarie Street, Sydney, by January 23, 1960, submitting copies of three original references.

Roussel Pharmaceuticals (Pty.) Ltd.

The Board of Roussel Pharmaceuticals (Pty.) Ltd. has announced the appointment of Mr. J. T. Hook as Managing Director. Mr. Hook was formerly General Manager of Roussel Pharmaceuticals (Pty.) Ltd. and in his new appointment will cover the Company's operations in Australasia.

The William Gibson Research Scholarship for Medical Women.

The Royal Society of Medicine invites applications for the William Gibson Research Scholarship from women who are British subjects, and who hold a registrable medical qualification.

The scholarship, which is normally awarded for two years, but which may be extended for a third year, is for £200 *per annum*. In choosing the scholar, the Council of the Society will be guided by the research work already

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED NOVEMBER 28, 1959.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.[2]	Australia.[2]
Acute Rheumatism	..	4(1)	..	..	..	..	..	..	4
Amœbiasis	..	..	..	..	1(1)	..	..	..	1
Ancylostomiasis	..	..	2	..	..	..	6	..	8
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1(1)	..	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2(2)	8(7)	2(2)	..	..	1	3	..	16
Diphtheria	..	..	1	..	4(4)	..	..	..	5
Dysentery (Bacillary)	..	..	..	1	2(2)	..	..	..	3
Encephalitis	1	..	..	1(1)	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	89(28)	49(32)	26(6)	11(7)	4(4)	..	1	..	180
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	1	..	..	..	..	..	..	..	1
Malaria	..	1(1)	..	1(1)	1(1)	..	..	..	3
Meningococcal Infection	1	1(1)	..	..	..	1	..	..	3
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1	..	..	..	..	..	..	..	1
Puerperal Fever	8(1)	..	..	..	..	..	..	..	8
Rubella	..	23(16)	..	..	1(1)	..	..	..	24
Salmonella Infection	..	..	..	2(2)	..	..	..	..	2
Scarlet Fever	6(3)	24(17)	..	10(5)	..	2(1)	..	..	42
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	1	..	124	..	125
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	21(14)	24(17)	8(6)	5(4)	11(4)	4(2)	..	..	73
Typhoid Fever	..	..	..	..	1	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

[2] Figures incomplete owing to absence of return from Australian Capital Territory.

done or contemplated by candidates. It is expected that a candidate will hold a recognized appointment, and that the scholarship will be a useful aid to research either in the United Kingdom or abroad.

There is no examination, nor need a thesis be prepared for publication; but Council expects that an annual report will be received from the scholar on work made possible by the award, and that due recognition of the award will be given in any papers subsequently published.

The next award will date from October 1, 1960, and applications must be received by the Society by May 1, 1960. They should give details of professional training received, appointments held and research work undertaken or contemplated. The names of two referees should be given. Applications should be addressed to the Secretary, Royal Society of Medicine, 1 Wimpole Street, London, W.1.

Australian British Canadian Travelling Fellowship, 1960.

The Australian Orthopædic Association has appointed Dr. W. G. Doig, orthopædic surgeon at Prince Henry's Hospital, Melbourne, as the Australian British Canadian Fellow to tour America in 1960.

The Frank H. Rowe Memorial Fellowship.

The Federal Council of the British Medical Association in Australia has announced that the Frank H. Rowe Memorial Fellowship for post-graduate training in physical medicine and rehabilitation has been awarded to Dr. D. A. Dowie, of Crafers, South Australia.

Post-Graduate Work.

ROYAL PRINCE ALFRED HOSPITAL: EAR, NOSE AND THROAT DEPARTMENT.

Seminar Programme, 1960.

THE staff of the ear, nose and throat department of the Royal Prince Alfred Hospital will conduct a seminar on the second Saturday of every month at 8 a.m. in the Scot Skirving Lecture Theatre. The main speaker will not exceed forty minutes, and there will be a discussion at the conclusion of his remarks. All medical practitioners and clinical students are invited to attend.

At the next seminar, to be held on January 9, 1960, Dr. J. H. Seymour will speak on "Cortical Deafness. Studies in the Physiology of Speech and Aphasia".

University Intelligence.

UNIVERSITY OF SYDNEY.

THE following staff changes in the University of Sydney have been announced.

Professor L. Dods, who has occupied the Chair of Child Health since 1949, has informed the Senate of his intention to resign from that position as from March, 1959.

Dr. D. C. Mackenzie has been appointed to a senior lectureship in surgery. Previously he was a research fellow in vascular surgery.

Dr. D. W. Piper, who was previously a part-time lecturer in medicine and in physiology, has been appointed to a senior lectureship in medicine.

Dr. M. G. Taylor, who was previously lecturer in physiology in the Medical School of St. Bartholomew's Hospital, London, has been appointed to a senior lectureship in physiology.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Lang, George Andrew, M.D., 1952 (Univ. Budapest) (Provisional Registration, Section 17 (1c)), 10c Challis Avenue, Potts Point.

Stockler, Gabor, M.D., 1953 (Univ. Budapest) (Provisional Registration, Section 17 (1c)), 143 Hall Street, Bondi.

Diary for the Month.

JANUARY 8.—Queensland Branch, B.M.A.: Council Meeting.
JANUARY 8.—Tasmanian Branch, B.M.A.: Branch Council.
JANUARY 12.—New South Wales Branch, B.M.A.: Council Quarterly.
JANUARY 18.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
JANUARY 19.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JANUARY 21.—Victorian Branch, B.M.A.: Executive of the Branch Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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